A REPORT ON THE USE OF DDT IN THE PROVINCE OF ONTARIO

Prepared by
THE ONTARIO PESTICIDES ADVISORY BOARD

ONTARIO DEPARTMENT OF HEALTH
HONOURABLE THOMAS L. WELLS, MINISTER

ONTARIO

A

REPORT

ON

THE USE OF

DDT

IN THE
PROVINCE OF
ONTARIO

Prepared by
THE ONTARIO PESTICIDES
ADVISORY BOARD

At the Request of
THE MINISTER OF HEALTH
ONTARIO DEPARTMENT OF HEALTH

September, 1969

Produced for the
PESTICIDES ADVISORY BOARD
by the
COMMUNICATIONS BRANCH
ONTARIO DEPARTMENT OF HEALTH

HO-42936 42-25-5501 1/70–1M

PESTICIDES ADVISORY BOARD

The Pesticides Advisory Board was established under the authority of *The Pesticides Act, 1967* to advise the Minister of Health on matters pertaining to the use of pesticides in the Province of Ontario.

The current membership of the Board is as follows:

H. E. Gray, B.A., B.Sc., M.Sc., Ph.D.
Chairman

G. S. Cooper, B.A., B.Sc., M.Sc., Ph.D.
B. E. Beeler, B.S.A., M.Sc.
*R. Frank, B.Sc., M.S.A., Ph.D.
A. Gartner
K. G. Laver, B.S.A.
F. Scott-Pearse, B.S.A.
K. B. Turner, B.Sc.F., M.Sc.F.,
D. W. Wilson, B.S.A.
M. C. Wood

Secretary to the Board
D. L. Bogaerts

*R. Frank, B.Sc., M.S.A., Ph.D. was an alternate for Mr. Beeler during this study.

ONTARIO

Pesticides Advisory Board

DR. H. E. GRAY
Chairman

ONE ST. CLAIR AVENUE WEST, TORONTO 7.

TELEPHONE 416 365-6636

D. L. BOGAERTS
Secretary

September 1969.

Hon. Thomas L. Wells,
Minister of Health
of the
Province of Ontario.

Dear Mr. Minister:

We, your Pesticides Advisory Board, respectfully submit to you our report on the matter of DDT which was referred to us on July 11th, 1969.

Obediently yours,

Harold E. Gray

Dr. Harold E. Gray
Chairman

HEG/mh

CONTENTS

APPENDICES

REPORT OF THE

PESTICIDES ADVISORY BOARD

SECTION A

Introduction

1. APPOINTMENT OF THE PESTICIDES ADVISORY BOARD

On March 1st, 1967, The Province of Ontario, through an Order-in-Council, established the Pesticides Advisory Board. The necessary legislation is contained in *The Pesticides Act, 1967,* which is administered by The Ontario Department of Health.

One of the duties of the Pesticides Advisory Board is to "consider and report upon any matter that may be referred to it by the Minister."

2. TERMS OF REFERENCE OF INVESTIGATION

On July 11th, 1969, the Pesticides Advisory Board was requested to investigate and report upon the current status of DDT* in Ontario, particularly in regard to its possible effects on human health and the environment; to consider its economic importance; and to make such recommendations as may be deemed necessary to current legislation.**

3. PROCEDURE

The amount of scientific and non-scientific literature which has been published on DDT is enormous. The study carried out by the

*DDT is defined in this report as technical DDT (p.p′–DDT and o,p′–DDT) and its metabolites (p,p′–DDD, also known as TDE, and p,p′–DDE).

**This study is the latest in a series of examinations of the DDT problem by the Ontario Government. The most recent previous study was the Interim Report on the Control of DDT Use in the Province of Ontario, issued March 1969 under the auspices of the Advisory Committee on Pollution Control.

Board included a review of some scientific literature but, because of the scope of the literature, reliance was placed primarily on interviews and consultation with specialists from across North America. The Board is deeply grateful to the many persons who, through their information, materially assisted in this study.

SECTION B

Background Information on DDT

(DDT — Dichloro-diphenyl-trichloroethane)

1. PHYSICAL, CHEMICAL AND BIOLOGICAL PROPERTIES OF DDT

Technical DDT contains at least 70% of the p,p′ isomer and most of the remainder is the o,p′ isomer. The pure p,p′ isomer is a fine crystalline powder with a melting point of 108.5° C, with a low vapour pressure of 1.9×10^{-7} mm Hg at 20°C. It is practically insoluble in water (1.2 parts per billion), moderately soluble in hydroxylic and polar solvents such as alcohol and in petroleum oils, readily soluble in most aromatic and chlorinated solvents. It is readily dehydrochlorinated when in solution in organic solvents by alkali or organic bases; otherwise it is stable and inert.

The acute oral LD_{50} of technical DDT to male white rats is 113 mg/kg. There is little evidence of chronic toxicity.

There is scientific evidence that DDT has a half-life in the soil of up to 12 years.

2. HISTORY OF DDT

DDT was originally synthesized in 1874, but its insecticidal properties were not discovered until 1939 by the Swiss chemist, Paul Mueller. For his work Mueller received the Nobel Prize in 1948. It was awarded because of the unprecedented contribution of DDT in saving millions of human lives from typhus, malaria and a number of other insect-borne diseases.

DDT was the miracle chemical of World War II and shortly afterwards became available for civilian use. Since then DDT has been used more widely throughout the world than any other

insecticide. A vast amount of literature has been built up on DDT over the years, some of which is conflicting or unscientific. Much of the recent scientific literature has indicated that DDT has caused environmental stresses in certain species of wildlife because of its persistence and its build-up in the food chain.

A major problem confronting scientists in the early years of DDT use was the need for a more accurate method of detection than the spectro-photometric procedure. A turning point came in 1963 with the introduction of gas liquid chromatography when it became possible to measure with a thousandfold increase in sensitivity. This breakthrough permitted more accurate research to be carried out and gave regulatory agencies a valuable tool to enforce legislation on food contamination.

3. HISTORY OF DDT USE IN ONTARIO

DDT was first used jointly by the Ontario Department of Agriculture and the Federal Department of Labour for the control of mosquitoes and house flies in labour camps in 1943. In 1944 the Department of Lands and Forests initiated experiments in the use of DDT for control of forest insects, particularly the spruce budworm which was devastating large sections of the province at that time. Because DDT was the first of the broad-spectrum contact insecticides, the Department was quick to recognize the potential of the chemical to cause widespread damage to other forms of life when applied in an over-all spray. Therefore, in the 1944 experiments and in the larger sprayings of 1945, the Department engaged the leading wildlife biologists available to investigate the impact of DDT on various forms of wildlife.

In the two major reports that were published on this work*, it was concluded that while there was considerable direct mortality of amphibians, reptiles and some species of aquatic life, with the high dosages of DDT believed necessary at that time, there was likely to be a good recovery of populations within a relatively short time. These early investigators were looking for direct toxicological effects only, and had no way of knowing that DDT could eventually have

*1. Kendeigh, S. Charles. Bird population studies in the coniferous forest biome during a spruce budworm outbreak. Biol. Bull. 1, Ontario Dept. Lands and Forests, Toronto 1947.

2. Ontario Dept. Lands and Forests. Forest spraying and some effects of DDT. Biol. Bull. 2, Toronto 1949.

serious indirect detrimental effects. Nevertheless, following the large-scale test of 1945 (64,000 acres), DDT has never been used on a large scale for forest spraying in Ontario, largely because the two major problems on an area basis, the spruce budworm and the forest tent caterpillar, did not justify the cost of control programs. In recent years, fear of possible side-effects of DDT became a major consideration.

In addition to the foregoing, many other general uses for DDT developed quickly in the post-war period. For example, the military, through its general knowledge of DDT, introduced mosquito and black fly control programs at military establishments throughout the country. Within a few years these programs were followed by several municipalities in South-Western Ontario and later by some Northern communities, including tourist camps and resort areas. DDT also became widely used by the home gardener. Of some significance from a pollution standpoint was the use of DDT by municipalities, private citizens and others to spray elm trees in an attempt to control the Dutch elm disease.

Following the conclusion of World War II, DDT was widely adopted by the agricultural industry to replace such insecticides as lead arsenate, nicotine sulphate, and paris green. Its wide spectrum of activity and much lower toxicity made its acceptance rapid. The first spray calendar recommendation by the Ontario Department of Agriculture appeared in 1947 for its use on many vegetables. It controlled the major pests of potatoes, the Colorado potato beetle, tarnished plant bug and aphids, as well as insects on peas, beans, corn and cole crops.

The following year it appeared on the spray calendar for fruit for use in apple orchards for the control of codling moth and apple maggot, the two most serious insect pests. For peaches it was recommended for the control of the Oriental fruit moth and other pests. DDT was also found to be effective for the control of soil insects such as wireworms and cutworms. The livestock industry found it to be effective for the control of flies in barns and on animals. In the period 1949-1954 its use in agriculture was almost universal.

Signs of resistance to DDT were detected in the early 1950's and very soon growers were forced to switch to alternatives for some pests. Aphids on such crops as potatoes were one of the first to show

this in the field and by 1955 malathion was used instead. Flies in barns showed resistance to DDT after only a few seasons of use and it was replaced by various organophosphates in the 1950's. The greater effectiveness of the cyclodienes, a sub-group of the organochlorines which includes aldrin, dieldrin, heptachlor, for the control of most soil insects resulted in less use of DDT by the late 1950's. Only the emergence of the darksided cutworm as a major pest in tobacco, not controlled by the cyclodienes, caused an almost complete return to the use of DDT for this particular crop.

The vegetable processing industry had used DDT widely for the control of aphids on peas, and cornborer and corn earworm on sweet corn. About 1958 it became evident that if corn stover or pea vines were treated with DDT and subsequently fed to livestock a definite residue problem resulted. The introduction of an effective alternative, carbaryl (Sevin), for corn enabled growers to discontinue the use of DDT by 1962. The use on peas was greatly reduced at the same time but did not cease entirely until 1964.

The introduction of the organic phosphates in the 1950's and of the carbamates in the 1960's, along with the development of resistance, has led to the decrease in the use of DDT in agriculture since about 1955. Only in tobacco production has there been a need for an increase in use.

With these wide uses of DDT, it was inevitable that there would be misuse of the material at times with respect to location, type of problem, amount applied, and methods of application.

4. HISTORY OF GOVERNMENT ACTION IN ONTARIO

The first evidence that DDT could pass from the diet of dairy cattle to milk came in research work reported in the United States in 1946. Between 1946 and 1952 further studies confirmed these findings and created a concern in the dairy and meat industries. In 1953, The Food and Drug Directorate of the Department of National Health and Welfare established official tolerances for all agricultural commodities. Most food commodities were permitted a maximum level of 7 ppm but with dairy products no residue was allowed.

About the same time, the Ontario Department of Health took the first official step to restrict the use of DDT in the province, which resulted in the following regulation of *The Public Health Act* of 1954:

(1) DDT and TDE shall not be used for extermination:*
(a) in such a way as to contaminate food or drink for human consumption; OR
(b) in concentrations of over 10 per cent in rooms used for human habitation.

In 1956, the Ontario Department of Health gave further recognition to the whole subject of pesticides by transferring these matters from *The Public Health Act* into a separate legislative program, *The Pesticides Act.* The restrictions on DDT remained as they were in 1954.

In 1957, as a result of finding dieldrin in dairy products in Western Canada, a survey was made throughout Canada for dieldrin and other chlorinated hydrocarbons. DDT was found generally with the result that joint federal and provincial recommendations were made to stop the use of DDT on animals and animal feeds (corn and peas).

In 1961 the use of DDT after the second cover spray for peaches was deleted from the spray calendar recommendations.

In 1963 a considerable amount of the DDT used in apple production was replaced by carbaryl (Sevin). In the same year DDT was deleted from the controls recommended in the spray calendar against armyworms.

In 1964 a further revision of *The Pesticides Act* provided that:

An exterminator shall not use aldrin, chlordane, dieldrin, DDT, TDE, endrin or lindane,

(a) in any building used by an animal that produces milk for human consumption or any milk room;

(b) on any pasture or forage being used or to be used by animals producing milk for human consumption; OR

(c) in such a manner as to come in contact with or likely to come in contact with food or drink for human consumption.

*Also known as DDD

The Ontario Water Resources Commission Act of 1956 provides that: "No person shall add any substance to the water of any well, lake, river, pond, spring, stream, reservoir or other watercourse for the purpose of killing or affecting plants, snails, insects, fish or other living matter or thing therein without a permit issued by the Commission." Since 1966 the Commission has not issued any permits for the use of DDT in water.

In 1966 the Department of Lands and Forests discontinued using DDT for control of mosquitoes and black flies, and at the end of the 1967 season discontinued the use of DDT entirely in all of its programs.

In 1967 further restrictions in the method of application of DDT were introduced into *The Pesticides Act* by the Department of Health. This legislation restricted the application of DDT by aircraft, concentrated air blast machine and power duster to a permit basis, which could be refused if the application could not be carried out safely. Furthermore, the DDT had to be applied in liquid or granular form and records of all such applications had to be kept by the applicator.

In 1967 the spray calendar recommendation for DDT for the control of corn borer on field corn was deleted. In the following year the use of DDT was minimized in the spray calendar and it was not recommended for many agricultural crops. The use on apples after July 20th was deleted, and DDT had already been replaced by carbaryl (Sevin) and azinphos-methyl (Guthion) in much of the spraying done before that time of year. Less persistent insecticides were recommended as a substitute for DDT on many vegetables. In the field crops there were no recommended uses except for tobacco.

In 1969 the use of DDT was deleted from the spray calendar recommendations for corn borer and corn earworm on corn, and for all insects generally on beans, cabbage, cauliflower, Brussels sprouts, lettuce, peas, peppers and tomatoes. In the case of peaches, all uses were deleted except for plant bugs.

*The Pesticides Act,** as amended in 1969, provides that:

Group C substances made up of (1) DDT; DDD; TDE; . . .

No person shall use any Group C substance in an extermination in such a manner that the substance comes in contact with an area other than the area to be treated.

An exterminator shall not use . . . DDT; TDE; . . .

(a) in any building used by an animal that produces milk for human consumption or any milk room;

(b) on any forage being used or to be used by animals producing milk for human consumption or being prepared for slaughter for human consumption; OR

(c) in such a manner as to come in contact or likely to come in contact with food or drink for human consumption.

*Editor's Note: The Ontario Regulation 386/69 under *The Pesticides Act, 1967,* relating to the restricted use of DDT and TDE after the 1st day of January, 1970, which came about as a result of this Report of the Pesticides Advisory Board, has been added as Appendix VIII.

SECTION C

Findings

1. PUBLIC HEALTH HAZARDS

The Board interviewed the following:

1. Dr. J. R. Brown–University of Toronto
2. Dr. Wayland J. Hayes, Jr.–Vanderbilt University
3. Mr. J. Karfilis–Pollution Probe
4. Dr. E. Mastromatteo–Ontario Department of Health
5. Dr. R. B. Sutherland–Ontario Department of Health

In the 25 years that DDT has been used, there have been no reported deaths associated with its manufacture, handling or use. Nor has any death ever been reported from DDT residue on food. This statement can be made for very few other insecticides.

From Dr. Hayes' presentation, it was learned that the range of DDT and its metabolites in human fat varies from 2 to 37 parts per million on a global basis, and levels in the general population of United States appear to be in the order of 10 to 12 parts per million. These levels have shown no appreciable change in the past decade. Dr. Brown reported that levels from Ontario are lower than 5 ppm.

In special studies conducted by Dr. Hayes with humans, no clinical symptoms were present where storage attained levels 10 to 20 times higher than those found in the general public, which would indicate that there is no hazard to health.

Neurological and hepatic symptoms have been described in the Russian literature among workers who have had an occupational exposure to DDT production for periods of over 10 years, but not among members of the general populace. These observations have not been substantiated by North American studies when exposures have occurred up to 19 years.

Tarjan and Kemeny in Hungary recently published two papers indicating the potential carcinogenicity of DDT. The first of these was carefully reviewed by the 1967 joint FAO/WHO meeting on Pesticide Residues. Dr. L. Tomatis, Chief, Unit of Chemical Carcinogenesis of WHO, stated that "on the basis of data available today there is no convincing evidence of the potential carcinogenicity of DDT for man." A large collaborative study has been set up by the International Agency for Research on Cancer involving three institutes. No results from this study will be available for at least a year.

During 1968 Dr. Sutherland carried out a comparative study between Norfolk County, an area with a high use pattern, and the City of London, an Ontario urban area, to ascertain if a correlation existed between the use of DDT and deaths from causes which might be associated with its use. No statistical significance was noted between the number of recorded deaths and those expected.

The Honourable John Munro, Minister of National Health and Welfare, on more than one occasion, has stated that there is no proof that DDT is a danger to the health of Canadians. Research by his Departmental staff has shown that the average Canadian intake is less than 5% of the level established as safe by the World Health Organization.

The Pesticides Advisory Board concludes that DDT and its metabolites do not, at this time, or in the foreseeable future, constitute a public health hazard in Ontario.

Following are some of the comments of persons interviewed:

Dr. John R. Brown, Head
Department of Physiological Hygiene
School of Hygiene
University of Toronto.

Dr. Brown stated that, as a result of analysis of human fat samples from persons who died in Toronto from natural or accidental causes in 1966, the mean value for DDT and its metabolites was 4.39 ppm. These concentrations, he pointed out, do not appear to have increased since 1959-1960, and do not appear to constitute a threat to health at this time. See Appendix I.

Dr. Wayland J. Hayes, Jr.
Professor of Biochemistry
School of Medicine
Vanderbilt University
Nashville, Tennessee
U. S. A.

Dr. Hayes presented a picture in depth of the toxicological aspects of the chlorinated hydrocarbons with special emphasis on DDT. He dealt with both acute and chronic toxic aspects from the human safety point of view. All of the effects of DDT are related to the dosage.

While stored in all tissues, DDT is stored mainly in the fatty tissues. With repeated doses, the amount in storage gradually increases until an equilibrium is reached, when the amount lost is equivalent to the amount absorbed.

Dr. Hayes discussed the comparative physiology of birds and humans and explained how metabolism of each differed. He pointed out that birds are capable of mobilizing their fat reserve at such a rapid rate as to permit lethal levels of DDT to pass to the brain. In humans the mobilization of fat is so much slower that even high levels of DDT in storage fat are not released fast enough to deliver lethal levels to the brain. An additional built-in safety factor is that the human liver is much more efficient in removing DDT from the blood stream than that of birds.

A study of workers heavily exposed to DDT for as much as 19 years failed to reveal any illness attributable to their exposure. Volunteers have been safely fed DDT at a dietary rate over 900 times greater than that of the general population, (35 mg per year in United States), for an 18-month period without showing any clinical symptoms.

DDT has a good safety record, although it has remarkable persistency in animal and ecological systems. When it is replaced by compounds that are stored for shorter periods there may be increased hazards to workers. Mere lack of persistence and low acute toxicity is not a complete guarantee of safety.

The full text of a paper delivered by Dr. Hayes earlier this year in Sweden at a National Conference on the status of DDT appears in Appendix II.

Mr. James Karfilis
Legal Council For Pollution Probe and GASP
Toronto.

Mr. Karfilis expressed great concern regarding the possible effects which might show up in the human population in subsequent generations because of the use of DDT. He felt that a much longer period of time than the 27 years DDT has been used would be necessary to determine its effects on human life.

Dr. R. B. Sutherland
Chief, Health Studies Service
Environmental Health Services Branch
Ontario Department of Health.

During 1968, approximately 40 per cent of all DDT sold in Ontario in quantities of 160 fluid ounces or 4 pounds or more was for application in Norfolk County. Comparative data for earlier years are not available, but it can be assumed that for some years this county has probably received relatively large proportions of the DDT sold.

No human cases of disease of any kind have been reported in the literature from chronic ingestion of DDT. Based upon animal experimentation, it has been suggested that injury to the liver and kidneys might be looked for in humans. In addition, one might consider the possibility of central nervous system effects.

The number of male and female deaths which would have been expected during 1960-62 and 1965-67 among residents of Norfolk County from gastro-enteritis, cirrhosis of the liver, chronic nephritis and infections of the kidney, were estimated from the provincial age-specific death rates and compared with the numbers of deaths from these causes which actually occurred in the years mentioned. No significant deviation from the normal incidence of these diseases was found.

As a further check, a similar comparison was made of recorded and expected mortality during the same three-year periods for males and females in the City of London, a community where we may assume that very little DDT has been applied. Again, no significant increase in mortality from these diseases was found.

In summary, there was no evidence of any increase in the number of deaths from the causes mentioned over the number that would normally have been expected among residents of Norfolk County. See Appendix III.

2. ENVIRONMENTAL HAZARDS

The Board interviewed the following:

Mr. A. E. Armstrong	Department of Lands and Forests
Dr. D. A. Chant	University of Toronto
Dr. C. D. Fowle	York University
Dr. R. Frank	Provincial Pesticide Residue Testing Laboratory
Dr. C. R. Harris	Canada Department of Agriculture
Dr. H. Hughes	University of Toronto
Mr. G. Mains	Pollution Probe
Mr. J. H. Neil	Ontario Water Resources Commission
Dr. K. Ronald	University of Guelph

In recent months there has been an increasing awareness by analytical chemists that residues of another group of chemicals, known collectively as polychlorinated biphenyls (PCB's), may be mistaken for DDT in an analysis for residues. The PCB's are plasticizers, with potential sources arising from a wide range of manufacturing activities. However, it must be assumed for the present that most of all previous residue data are valid, and therefore on a global scale DDT has been found as a contaminant in air, soil and water. In the future it may be necessary to review the inter-actions of PCB's and organo-chlorine compounds.

Air pollution studies have not been made in Ontario, but work from other parts of the world suggests that minute quantities of DDT can be air-borne over a long distance.

DDT residues in Ontario agricultural soils vary from a trace to a maximum average of 60 parts per million in isolated pockets, i.e. apple orchards. Overall, the agricultural lands of Ontario carry less than 2 ppm. These levels do not appear to have any detrimental effect, and in fact, according to Dr. Harris, may be beneficial. DDT is

tightly bound in the soil and does not move into the ground water. However, there are studies to show that some contamination of the water environment may occur by wind and sheet erosion.

The somewhat limited number of samples taken from the agricultural areas of southwestern Ontario showed that DDT residues in waters vary from a non-detectable level up to about 0.08 parts per billion (0.00008 ppm). The average of these samples contained less than 0.02 parts per billion (0.00002 ppm). Only the illegal application of DDT directly to water will result in levels greater than the foregoing.

The real concern of ecologists is that low level contamination by DDT of the physical parts of the environment may lead to its concentration in certain organisms.

From the evidence available in the Western Hemisphere, the species that have caused most concern to date are:

(a) certain fish-eating birds, and

(b) certain predatory fish.

These two forms of wildlife constitute those members that feed either at or near the peak of the food chain where the concentration of DDT would be the highest. The high levels of DDT in these species have been reported by wildlife biologists throughout the world to:

(a) affect liver enzymes in birds and cause the laying of thin-shelled eggs, hence indirectly affecting reproduction, and

(b) affect the hatchability of fish eggs and survival of fish fry.

Studies are currently being carried out by the Ontario Government to determine the extent of this condition in birds, and to expand the present knowledge in fish. There is not yet any real evidence that DDT has been detrimental to the bird life of the province.

There is, however, strong evidence that DDT has affected the reproduction of lake trout in two locations in Ontario, namely, Lake Simcoe and the Muskoka Lakes. There are obvious explanations for the high DDT levels in these waters. Both areas are intensively developed as resort and recreational areas, and cottagers and other groups have had a long-standing annual program of spraying for control of mosquitoes and black flies. Additionally, the Muskoka area is plagued with periodic outbreaks of the forest tent caterpillar,

the last being from 1962-66, and DDT was used by the private groups for this problem as well. Most spraying for these problems has been by aircraft. The Muskoka region is a prime example of a non-agriculture area where, because of geological features, DDT would have a maximum opportunity to get into water even when applied over land. Exposed bedrock is common, soils are often thin, and consequently surface run-off is accentuated. In the case of Lake Simcoe, one must also recognize the intensive agricultural industry of the Holland Marsh as a potential source of DDT.

There is by no means any evidence that DDT has caused any general disturbance to the Ontario ecosystem. Other forms of pollution, stress and habitat destruction, which have been introduced by human activities, cannot be overlooked as major factors detracting from the well-being and survival of many species of fish and wildlife.

Following are some of the comments of persons interviewed:

Mr. A. E. Armstrong
Fish and Wildlife Branch
Ontario Department of Lands and Forests
Toronto.

Mr. Armstrong has co-ordinated the work of fish collections from provincial lakes and streams by his department and the submission of these to the Ontario Water Resources Commission and the Provincial Pesticide Laboratory for analysis. He presented a paper entitled "Report on Background Data on Use of DDT and Its Adverse Effect on Fish and Wildlife," and elaborated on those aspects that pertain to conditions in Ontario (Appendix IV).

He drew special attention to the lack of natural reproduction of lake trout in the Muskoka Lakes and correlated this condition with the fact that lake trout from these lakes had the highest levels of DDT of any fish from provincial waters. He discussed the current thoughts on fish fry mortality, due to DDT residues, and pointed out that losses appear to be associated with the absorption of the yolk sac.

In conclusion, he felt that monitoring should be continued and steps taken to curtail, and eventually eliminate, the use of DDT where it is likely to be detrimental to fish and wildlife, either directly or indirectly.

Dr. D. A. Chant
Chairman, Department of Zoology
University of Toronto
Toronto.

Dr. Chant was sent an invitation by registered mail to appear before the Board to give his views on the future use of DDT in Ontario. Because he was out of the province in the far north at the time of the meetings, he was unable to attend.

Dr. C. D. Fowle
Chairman, Department of Biology
York University
Toronto.

Dr. Fowle has done extensive research into bird populations in the New Brunswick forests following intermittent spraying with DDT for spruce budworm control. The New Brunswick studies have not delineated any significant problems on birds. Populations shifted according to the available food supply, and following an application of DDT, the availability of food was reflected in lower populations. As food supplies became available bird populations returned to normal.

While noting that DDT is an incredibly useful material, Dr. Fowle went on to point out some of its generally recognized adverse effects. For example, it concentrates in fatty tissue at levels which appear to follow a tenfold increase at each trophic level in the food chain. The toxicological significance of this is not clearly understood, although recent work has shed some light to explain the reproduction problem in some fish-eating birds. DDT inhibits the liver enzymes which regulate the deposition of calcium, thus resulting in thin-shelled eggs. There is no evidence that birds in general are exposed to sufficient DDT to be affected in this way. Dr. Fowle also referred to the work of Dr. John Anderson of the Fisheries Research Board of Canada, who has found that under laboratory conditions DDT makes young fish more susceptible to death from changes in water temperature, and also affects their ability to learn.

Dr. Fowle concluded that care is needed and restrictions should be placed on the use of DDT. However, caution is needed in the substitute organo-phosphorus insecticides, because these are often lethal to some wildlife in a more dramatic way.

Dr. R. Frank
Director, Provincial Pesticide Residue Testing Laboratory
Ontario Department of Agriculture and Food
Guelph.

Dr. Frank compared the levels of DDT and its metabolites found in samples from both agriculture and the environment in the following table:

DDT and METABOLITES

Sample	*No. of Samples*	*% Fat*	*In Whole Sample (ppm)*	*In Fat (ppm)*	*Remarks*
Water	45	—	0.00002	—	Southern Ontario
Soil	36	—	3.68	—	7 orchard and vegetable samples averaged 15.9 ppm. 29 other agricultural soils contained 0.73 ppm.
Milk	1651	4.0	0.005	0.134	All dairy producers in the province.
Milk – human	10	3.0	0.167	5.56	
Animal fats	66	100	—	0.25	Beef, pork, chicken fat 49 9 8
Pheasant —brains	125	—	0.09	—	Pheasants from Southern Ontario.
—fat	29	100	—	0.82	
Fish muscle	362	3.2	3.12	97.0	Includes Great Lakes and Ontario lakes and streams.
Bird eggs —grain eating	54	16.6	0.07	0.39	Migratory birds from Northern Ontario.
—fish eating	19	8.3	12.5	150.0	

It has recently come to light that materials known as polychlorinated biphenyls (PCB's) are present in wildlife tissues and are confused with DDT and its metabolites in analytical procedures. These compounds, which are derived from industrial products, are toxic to animal life. Dr. Frank was positive that those fish analysed by his laboratory from Ontario waters contained insignificant quantities of polychlorinated biphenyls and that the residues reported were in fact DDT and its metabolites. However, PCB's are being found in some species of waterfowl in Ontario.

He drew the following conclusions:
(1) milk, meat and pheasants from southern Ontario have relatively low background levels of DDT,
(2) fish from either the Great Lakes or inland lakes have relatively high residues, with the Muskoka Lakes having the highest levels, and
(3) eggs from migratory birds that feed in the U.S.A. and nest in northern Ontario have low levels of DDT where birds are grain-feeding, but high levels where birds are fish-eating.

A report on DDT residues in Ontario milk supplies is given in Appendix V.

Dr. C. R. Harris
Head, Pesticides Section
Canada Department of Agriculture
London.

Dr. Harris has been responsible for carrying out surveys on the DDT levels in agricultural soils of southern Ontario in the past few years. He stated that orchard soils have the highest levels of DDT residue, with 120 ppm, but that these are only small pockets in the total agricultural land mass. Vegetable and tobacco soils are next with 2 to 6 ppm. In general agricultural lands have quite low residues. He said that no soils have reached a saturation level and that this might well be many times the highest residue reported. He also stated that the levels so far found are not biologically active. Because of the low solubility of DDT, and the fact that it is strongly held in the soil, no DDT has been found in drainage water and hence movement into water occurs only as a result of wind and water erosion or spray drift.

Dr. Harris reported that DDT has had no effect on earthworms even though high residues could be found in their adipose tissues. In the soil, DDT appears to be beneficial to collembola, a soil insect responsible for the decomposition of organic matter. DDT has not been detrimental to soil micro-organisms but fear was expressed that the substitute organo-phosphorus insecticides might well be detrimental and could ultimately affect soil fertility.

Dr. H. Hughes
Department of Physiological Hygiene
School of Hygiene
University of Toronto
Toronto.

Dr. Hughes has carried out a number of analyses on the tissue of sea gulls and had what he at first thought were high levels of DDT. Later he was convinced that these must be erroneous and concluded that the high values must have been due to the presence of polychlorinated biphenyls.

He also wondered whether the same might be true for some of the high values found in fish from the Muskoka Lakes.

Mr. G. Mains
A Student Representing Pollution Probe
University of Toronto
Toronto.

Mr. Mains presented the following papers written by Dr. C. F. Wurster, State University of New York.

1969 (1) DDT reduces photosynthesis by marine phytoplankton – Science 159:1474
(2) Chlorinated hydrocarbon insecticides and the world ecosystem – Biological conservation.
(3) DDT renders a declining reproduction in the Bermuda petrel – Science 159:979.

Mr. Mains built his own comments around these papers but, having no personal experience, could not contribute beyond the scope of the papers.

Mr. J. H. Neil
Director, Division of Laboratories
Ontario Water Resources Commission.

Mr. Neil discussed the level of DDT in fish of Ontario waters, and pointed out that the concentration of DDT in lake trout in the Muskoka Lakes has prevented reproduction. Maintenance of population is dependent on stocking by the Department of Lands and Forests. In comparing the data on DDT levels in fish from the Great Lakes, he thought that the level in Lake Erie was the lowest because the fish being caught there were the youngest. In explaining this he pointed out that as a fish increases in age, its level of DDT per gram of tissue also increases.

Mr. Neil stressed the need for more information on the effects of DDT on hatchability of fish eggs and survival of the young immediately after hatching.

Dr. K. Ronald
Chairman, Department of Zoology
University of Guelph
Guelph.

Dr. Ronald has done extensive research on marine life. His general philosophy was that the balance of nature should be permitted to return by removing not only DDT but all pesticides. He went even further and stated that man had over-reached himself and that defective human beings, in his opinion, should not be permitted to reproduce. Dr. Ronald appeared to be not overly concerned about DDT, but was more concerned about pesticides of low toxity including herbicides. These compounds, he felt, were a greater potential menace to wildlife in that they were more likely to change behavioural patterns.

Commenting upon the paper published by Dr. Wurster, which held that DDT could reduce photosynthesis in marine phytoplankton, Dr. Ronald stated that, with the slaughter and virtual extinction of whales in the Southern Seas, phytoplankton was so abundant that controls are needed to regulate its growth. Dr. Ronald drew attention to the fact that the many ships sunk during World War II that contained DDT must have contributed to the DDT residues found in Antarctic penguins.

3. ECONOMICS

The Board interviewed the following:

Mr. G. Arnum	Plant Protection Division, Canada Department of Agriculture
Mr. George DeMeyere	Ontario Flue-Cured Tobacco Growers' Marketing Board
Professor H. W. Goble	University of Guelph
Dr. C. R. Harris	Canada Department of Agriculture
Dr. W. O. Haufe	Canada Department of Agriculture
Dr. E. W. McEwan	University of Guelph
Dr. W. B. Mountain	Canada Department of Agriculture
Mr. P. G. Newell	Ontario Flue-Cured Tobacco Growers' Marketing Board
Mr. J. A. Oakley	Canadian Agricultural Chemicals Association
Dr. J. H. Phillips	Canada Department of Agriculture
Mr. B. P. Richardson	Ontario Pest Control Operators Association
Mr. H. Trotter	Canada Manufacturers of Chemical Specialties Association
Mr. L. Vickery	Canada Department of Agriculture
Mr. R. Wight	Plant Products Division, Canada Department of Agriculture
Mr. B. Wilson	Ontario Fruit and Vegetable Growers' Association

DDT no longer plays a major insecticidal role in the Province of Ontario. Over the past 15 years its use has been reduced by insect resistance and the introduction of equally or more effective chemicals which are less persistent. Newer materials have been accepted even though their cost is usually much higher; their short-term action usually necessitates more frequent applications, which result in repeated human exposure.

In forestry, DDT is no longer used in Ontario and any change in the regulations governing its use will have no economic or any other effect. In a recent publication of the Department of Agriculture and Food on the control of insects on ornamental trees and shrubs, DDT was replaced entirely by other insecticides. A small amount of DDT is still being used, for economic reasons, for the control of the elm bark beetle.

For the control of biting flies, mosquitoes and black flies, there are very satisfactory alternatives available at very little increased cost. These newer materials have been studied by the Biology Branch of the Ontario Water Resources Commission to ensure that they will not be a hazard to many other organisms in the water environment.

The Structural Pest Control Association feels that there is no satisfactory alternative to DDT for the control of bats and as a tracking powder for the control of mice in buildings. Any alternative chemicals would pose a far greater health hazard and mechanical methods are either too slow or prohibitively expensive (Appendix V).

In agriculture there remains a number of large or small problem areas where there is no substitute at present for DDT. Tobacco growers must presently use two applications of DDT at one and four pounds per acre to control all species of cutworms. Although research is almost complete on at least two alternative insecticides, neither will be available in more than token quantities until at least 1971. For the 1970 season, therefore, these growers will have to use DDT if they are going to grow tobacco. It has been agreed by the committee responsible for recommending tobacco insecticide programs and by the Ontario Tobacco Marketing Board that for one season it will be acceptable to use only a single application of approximately one pound of DDT per acre. This will result in an 80% reduction in the amount applied. It is hoped to discontinue even this use in 1971, if the alternate insecticides are available in sufficient quantity. The cost of these alternatives may be as much as ten times that of DDT, but the growers appear to be willing to accept this.

Fruit growers may have a problem in replacing DDT in a few of their spray programs. Plant bugs are a sporadic but serious pest of apples and peaches. At present DDT is the only chemical recommended for their control. This is not to say that none of the newer materials are effective, but rather that no research has been done to prove their effectiveness and therefore there are no label recommendations to allow them to be used. This same situation is true for the cane fruits, raspberries and black berries, where there is no alternative for the control of the caneborer. DDT is the only insecticide that will control the darksided cutworm on vegetable crops such as tomatoes, peppers and egg plant. The small acreage of these crops has not justified research time, residue studies and labelling requirements.

Livestock producers have almost completely eliminated the use of DDT in Ontario. In Western Canada, problems still exist where there is no satisfactory alternative for DDT for horn fly control on beef cattle on range and for blowfly on sheep. In Ontario, only the northern beef cattle operations may have need of DDT.

Concern was expressed by the representatives of the fruit and vegetable growers that the use of more costly alternative programs to DDT will put the Ontario producer at a disadvantage in selling his product in the home or export market in competition with growers from other provinces or countries. It was also pointed out that if there is any health hazard from DDT it is rather pointless to discontinue its use in Ontario if imported foods contained DDT residues.

The Board is concerned with the differences which appear between the uses for DDT which are permitted by the Federal Department of Agriculture under *The Pest Control Products Act* and those which may be permitted by this Province under *The Pesticides Act.*

Following are some of the comments of persons interviewed:

Mr. G. Arnum
Regional Supervisor
Canada Department of Agriculture
Plant Protection Division
Toronto.

Mr. Arnum is responsible for the plant quarantine and certification programs in Ontario. He indicated that DDT is not being used or recommended in any of their primary programs. However, it is one of the suggested pesticides in such secondary programs as potato certification.

For certain eradication programs, such as Japanese beetle and European chafer eradication, the banning of the use of aldrin and dieldrin has forced reconsideration of DDT even though it is less effective. The Plant Protection Division urged that they be authorized to continue using dieldrin under Section 22a (2b) where they perform the work themselves.

Mr. George DeMeyere
Chairman, Ontario Flue-Cured Tobacco Growers' Marketing Board
Tillsonburg.

Mr. DeMeyere indicated the Tobacco Marketing Board wished to switch to alternative pesticides as soon as possible. He gave two reasons. First, the exporting customer does not want DDT residue on the tobacco. Secondly, because of considerable public pressure against smoking in Canada, the use of DDT serves to aggravate this situation.

He added that at present there is no suitable alternative control and that is why the tobacco growers have not changed from using DDT.

He agreed to have his Board assist in any controlled sales program.

Professor H. W. Goble
Provincial Entomologist
Department of Zoology
University of Guelph
Guelph.

Professor Goble expressed the view that DDT has a place as an agricultural insecticide at present. He felt, however, that in time it will be replaced by newer insecticides. He expressed concern about the lack of knowledge of these newer materials. Many replacements have a short residual life but a higher mammalian toxicity resulting in a greater handling hazard. Others are less effective than the materials they replace, e.g. chlordane for aldrin and dieldrin.

He stated that DDT was still being recommended in the Ontario Spray Calendars for a number of uses, particularly the control of plant bugs on apples and other fruits, because no satisfactory alternatives are known. DDT recommendations have been removed from the new publication, *Insects of Trees and Shrubs.* O.D.A.F. Publication 93.

Dr. C. R. Harris
Head, Soil Pesticide Behaviour Section
Canada Department of Agriculture
London.

Dr. Harris, who is conducting much of the research into the replacements for DDT for the control of soil insects in the province, stated that suitable replacements are available for most soil uses. Three possible alternatives, Dursban, Bayer 37289, and Stauffer N-2596, all organo-phosphorous, are under intensive research for cutworm control in tobacco. Dursban and Bayer 37289 are in advanced stages of development and a limited quantity of Dursban will be available for the 1970 season.

The materials are more acutely toxic than DDT but, because they have only a very short residual life in the soil, their use may result in poor control under certain conditions. The cost of these new products will be several times that of DDT.

Dr. Harris was not anxious to see DDT banned for several reasons. He felt that DDT was not a large contributor to water pollution, since it is tightly held in the soil. The persistence of some possible replacement pesticides may be as long as DDT. Dr. Harris also felt that the use of DDT in tobacco production could be significantly reduced by revising the recommended practices for the next season or two.

Dursban:	O,O-diethyl O–(3,5,6–trichloro–2–pyridyl) phosphorothioate. LD_{50}–acute oral 97–276 mg/kg for rats, 1000–2000 mg/kg for rabbits. Registered in Canada, 1969, for mosquito control.
Bayer: 37289	O–ethyl O–2,4,5–trichlorophenyl ethyl phosphonothioate. LD_{50} – acute oral 16–35 mg/kg for rats. Not yet registered.
Stauffer: N-2596	S–(p-chlorophenyl) O–ethyl ethane phosphonodithioate. LD_{50} – acute oral 6 mg/kg for rats. Not yet registered.

Dr. W. O. Haufe
Officer-in-Charge
Livestock Entomology Section
Canada Department of Agriculture
Lethbridge, Alberta.

Dr. Haufe stated that DDT was not replaceable for horn fly control on beef cattle on western range. He added that the lack of control could result in 20–24% loss in rate of gain.

Dr. Haufe mentioned the need for DDT to control blowfly strike on sheep and tick control on range cattle. He stated that he foresees no immediate new pesticides for these purposes.

Dr. Haufe noted that he was not familiar with the Eastern Canadian requirements and, therefore, his comments had to be restricted to western conditions. He noted that no one within his Service was working on these problems in the East.

Dr. E. W. McEwan
Department of Zoology
University of Guelph
Guelph.

Dr. McEwan stated that there were substitutes for all vegetable and fruit uses for DDT but that many, such as parathion for plant bug control, were deadly poisonous to the user and had resulted in a number of cases of hospitalization of the users.

He felt that it would be a mistake to ban the use of DDT since he was unaware of any data showing it to be a public health hazard.

He outlined his experience with DDT in New York State, particularly the lack of adverse effects to fish and wildlife following its repeated use over a period of years on grapes in the Finger Lakes area.

Dr. William B. Mountain
Director, Vineland Research Station
Canada Department of Agriculture
Vineland.

On Friday July 11th, 1969, the Chairman had a telephone conversation with Dr. Mountain, who was unable to attend the meetings held by the Board on the use of DDT.

Dr. Mountain felt it would be regrettable to ban its use, but felt it should be strictly regulated in Ontario and used for food production by growers only. He considered it one of the safest insecticides for the grower to use, that it was essential for cutworm control, and felt it had greatly reduced crop losses compared to the materials it had replaced. This was particularly true in the case of apples where the loss was about 25% when lead arsenate was used, while with DDT the loss was essentially zero.

He felt that the problem with DDT had arisen largely through misuse by home-owner and cottagers who had used excessive amounts to control garden pests and biting flies.

In his opinion, if DDT is handled safely and sanely, it still has an important role to play in Ontario Agriculture.

Mr. P. G. Newell
Director, Ontario Flue-Cured
Tobacco Growers' Marketing Board
Newcastle.

Mr. Newell, who is a leading tobacco grower, described the present recommended practices for using DDT for cutworm control and how they have developed. Ten years ago aldrin and heptachlor were commonly used for cutworm control since the population then consisted of the black and variegated species with very few of the dark-sided species. However, continued use of these pesticides resulted in a major population change in favour of the dark-sided cutworm which was not controlled. DDT did control this species and a change in the recommendations was made in 1966. This involved the use of one pound of DDT on the rye cover crop immediately before it was ploughed down in the spring, followed by four pounds applied to the bare soil at least one week before planting. This program has successfully controlled all three species.

He agreed with Mr. DeMeyere that the growers would accept a substitute if it were available. The new products that will become available were discussed, including their effectiveness, hazard and the increased costs.

Mr. J. A. Oakley
Canadian Agricultural Chemicals Association.

Mr. Oakley stated that the C.A.C.A. members stood by their brief to the Advisory Committee on Pollution. He indicated that the demand for and the use of DDT are on the decline. But he also felt that there were several agricultural uses of DDT that were presently irreplaceable. These included the use of DDT for cutworm control in crops.

He noted that stocks of DDT for the 1970 season are already packaged and in the warehouse. It takes at least a year lead time to change formulations in the household market. He asked that there be a time period before legislation came into effect since the disposal by dumping of existing stocks might cause greater problems than their normal use.

He stated that industry was very concerned with the adverse publicity over the use of DDT. Some companies have already replaced it in all their small package lines. The association has no objection to its withdrawal from all uses in the home, in gardens or on pets. However, the members were against the principle of an outright ban on any pesticide.

Dr. J. H. Phillips
Vineland Research Station
Canada Department of Agriculture
Vineland.

Dr. Phillips expressed the opinion that orchard growers can get by without using DDT. Alternative chemicals or alternative programs will control the common pests which harm apples, peaches and grapes.

He was concerned that the use of DDT in the orchard is contributing to the environmental pollution problem since only about one-third of the applied chemical can be accounted for on the crop or on the soil. He felt that any use permitted in the future must not contribute to the general problem.

Mr. B. P. Richardson
Legal Committee
Ontario Pest Control Operators Association
Toronto.

Mr. Richardson submitted the attached brief on behalf of his association. The uses of DDT that cannot be replaced are as a tracking powder for mouse control and as a dust for bat control. With these two exceptions, he felt the structural pest control industry could operate without DDT. See Appendix VI.

Mr. H. Trotter
Canadian Manufacturers of Chemical Specialties Association.

Mr. Trotter stated that DDT is now being replaced in many small package lines and he felt that most would soon be DDT-free. The problem of replacement materials does not seem to be of concern and the added cost will just have to be included.

Mr. Trotter stated that most firms have already ordered containers for 1970 sales and that if DDT were banned the loss to them would be substantial. As a solution to this problem he suggested that an adequate time period be allowed to use up existing stock.

Mr. L. Vickery
Director, Research Station
Canada Department of Agriculture
Delhi.

Mr. Vickery outlined the research work carried out at the Tobacco Research Station. He corroborated Dr. Harris' findings and felt that no alternate to DDT would be available in sufficient quantities for the 1970 season. He also mentioned some of the difficulties involved in the use of these new products that might increase the cost of the new programs. For example, Dursban must be worked into the soil, whereas DDT only needs to be sprayed on to the soil surface.

He suggested for 1970 that the use of DDT should be modified by:

(a) making a single application of one pound per acre on the rye cover crop before it is ploughed instead of a total of five pounds per acre, one pound on the rye and four pounds to the soil before planting.

(b) not permitting any aerial application.

(c) not permitting the use of DDT for cutworm control on plants after they have been planted out on the field.

This single application may only be effective for a very few seasons since it will not control the black or variegated cutworms, which may re-emerge as the problem species.

Mr. R. Wight
Regional Supervisor
Plant Products Division
Canada Department of Agriculture
Toronto.

Mr. Wight outlined the responsibilities of the Plant Products Division under *The Pest Control Products Act.* He pointed out that not only were they concerned with effectiveness but also with crop residues. He referred to their recent proposals to limit some of the registered uses for DDT, as outlined in their letter T-26 dated June 11, 1969 (see Appendix VII). However, these would still leave DDT for use on a wide variety of pests on crops, livestock and for domestic use. He also said that most of these registrations would not be changed since they are used in other provinces.

Mr. B. Wilson
Representative, Ontario Fruit and
Vegetables Growers' Association
Toronto.

Mr. Wilson felt that the fruit and vegetable growers could protect their crops without the use of DDT with a very few exceptions. The pests mentioned were plant bugs on apples, cutworm in corn and flea beetle control on some vegetable crops. Less and less DDT is being used by growers each year.

The hazards of using some of the alternatives were of concern, as well as the increase in costs to the Ontario producer. Mr. Wilson felt that if Ontario banned the use of DDT it would put the producers in this province at a disadvantage.

Since Ontario is a large importer of fruits and vegetables, an increase in imports, which may carry high DDT residues, could increase the present dietary intake of DDT. This residue on imports should be controlled by border inspection and sampling.

4. DISPOSAL OF STOCKS OF DDT AND MATERIALS CONTAINING DDT

The Problem

The reason the Board suggested that the recommended changes "become effective within six months of enactment of the necessary legislation" is to provide sufficient time for those having DDT preparations in their possession to dispose of these materials legally. Many fruit and vegetable growers and farmers have a stock of DDT. It is not expected that they will voluntarily donate it for disposal by the Government. In the case of apple growers, a legal future use exists. A possible legal future use by the others exists under special permission. The Board recommends that the Department of Health undertake to establish quickly a reasonable policy in regard to the DDT held by farmers and communicate it to them forthwith.

Physical Disposal of DDT Stocks

Disposal of stocks of DDT can be accomplished in the following ways, listed by order of preference:

(a) by high temperature incineration, i.e. thermal degradation.
(b) through recommended agricultural uses.
(c) by burying the material in designated areas.
(d) by redistribution.

The destruction of DDT by high temperature incineration is the preferred method and the most expensive. This operation requires intense heat and would involve the use of a special type of furnace to break down the DDT. Unfortunately there are none of these in Ontario at the present time. The cost of the installations would limit the number of such incinerators and the cost of transportation would be considerable if any of these were available.

The second method is probably the most practical because the material can be distributed over a wide area and, if recommended agricultural uses are observed, there would be little damage to the environment. Furthermore, it would be by far the cheapest. However, it does not appear as if it will be possible to do this.

The third method would involve the choice of suitable sites in which stocks could be buried under Government supervision. Some costs would be involved, particularly those of transportation.

The fourth method would be to collect and donate the suitable formulations to WHO for use in their World Health programs.

SECTION D

Summary and Conclusions

This report has dealt with various facets of a many-sided problem. The evidence and conclusions have already been recorded in detail in the body of the report. This summary, therefore, is presented in the form of a series of brief statements.

(a) There was no conclusive evidence presented by any of the witnesses who appeared before the Board, or in any of the scientific literature reviewed, that indicates that DDT poses a threat to human health at this time.

(b) Information was presented on the long persistence of DDT and its entry into the food chain. It was largely on the basis of this information that the Board made the recommendations to reduce drastically the use of DDT.

(c) Evidence was presented which indicated that DDT adversely affects fish-eating birds and predatory fish.

(d) The Board recognizes that substitute compounds may cost some of the food producers several times the cost of DDT. Consequently, if produce which has been treated with DDT is allowed entry into the province, the Ontario producer will be placed at a considerable economic disadvantage.

(e) In general, it is the conclusion of the Board that the introduction of any persistent pesticide into the environment should be avoided when possible.

SECTION E

Recommendations

1. SUGGESTED LEGISLATION

That the use of DDT should be discontinued, except as follows:

1. under prescription of a qualified medical practitioner.
2. by a licenced structural exterminator for mouse and bat control.
3. for cutworm control in tobacco production.
4. for plant bug control in apple production.
5. when deemed advisable by The Minister to control emergency outbreaks of pests, e.g. hornfly control on beef cattle on range pasture.

That all sales of DDT be controlled in the following manner:

1. upon application, tobacco growers be issued a "buyer's permit" by the Department of Health which will permit them to purchase a definite amount of DDT. The amount would be based on the acreage allotted by the Ontario Tobacco Marketing Board.
2. upon application, apple growers be issued a "buyer's permit" by the Department of Health or the Department of Agriculture and Food provided that the local Fruit Specialist of ODAF certifies that a plant bug problem exists. The amount would be based on the acreage to be treated at the recommended rate.
3. upon application, the Department of Health may grant a "buyer's permit" for uses other than tobacco or apple production.

That, where DDT is to be applied by aircraft or concentrated air blast machine, a permit must be obtained from the Department of Health.

That these changes become effective six months from the time of enactment of the necessary legislation.

That these exemptions and restrictions be reviewed annually by the Board.

2. EXPLANATION OF RECOMMENDATIONS

It is the opinion of the Board that the use of DDT should be discontinued for a number of purposes which have contributed in the past to environmental pollution. These include its use in the home and garden, mosquito and biting fly control, and protection of shade trees. For home and garden use, DDT has been available as dusts, sprays and aerosols. In the hands of the average citizen, these materials are very often used improperly and at excessive rates resulting in residues of DDT in the population and the environment.

Although DDT has been used for mosquito, black fly and other biting insect control on a declining scale since 1966, it is prudent to stop all such uses. They have contributed in the past to the most serious instances of environmental pollution in the province.

The use of DDT on shade trees and plantations is unnecessary and a potential source of pollution, and should now cease completely.

The structural pest control industry has few uses for DDT nowadays and these uses should now be limited to those two for which no practical substitute is available – mouse and bat control. Neither of these two uses is likely to result in either large scale use or a contamination problem.

The agricultural industry has presently many potential uses for DDT, as evidenced by the current registered uses under *The Pest Control Products Act.* Many of these appear to be seldom used by the majority of growers because better alternatives are available. However, it is recognized that there are at least two problems for which no alternatives are available – cutworm in tobacco and plant bugs in apple production. The Board has, therefore, recommended that these uses be specifically excluded from any restriction on the use of DDT until such a time as satisfactory alternatives are available.

The Board is concerned that there may arise additional specific pest problems for which DDT is the only recognized method of control. A means must be provided whereby, after due consideration, the use of DDT may be permitted. It has been recommended that The Minister be empowered to designate such conditions. It is suggested that the Board, or some other representative group, should advise The Minister on such matters.

The control of the future use of DDT is most easily accomplished by ensuring that only those who need to use it are able to purchase it. Consequently, it has been recommended that users should apply to the Department for a permit to buy DDT. This permit would allow them to purchase a specific quantity which would be based on the acreage of tobacco or other crop to be treated. For the tobacco growers, the largest potential users of DDT, this acreage can be readily obtained with the co-operation of the Tobacco Growers' Marketing Board. In the case of other crops, it is recommended that the specialists of the Ontario Department of Agriculture and Food are the proper authority for assessing the requirements.

It is the opinion of the Board that special control procedures be exercised when DDT is applied by aircraft or concentrated airblast machine, since these types of equipment both have high potential for causing air contamination and drift deposits. Of the two, the concentrated airblast machine appears to cause the greater concern.

It is felt by the Board that a short period of adjustment is desirable in which DDT stocks can be disposed of in a safe and economic manner.

As soon as acceptable alternative insecticides become available, certain of the suggested exemptions could be deleted. It is anticipated that DDT for cutworm control in tobacco can be replaced by the 1971 season. This should be reviewed annually by the Board, or more frequently if the situation warrants it.*

*Editor's Note: The Ontario Regulation 386/69 under *The Pesticides Act, 1967*, relating to the restricted use of DDT and TDE after the 1st day of January, 1970, which came about as a result of this Report of the Pesticides Advisory Board, has been added as Appendix VIII.

APPENDICES

APPENDIX I

Reprinted from
THE CANADIAN MEDICAL ASSOCIATION JOURNAL
97,367-373, August 19, 1967

Organo-Chlorine Pesticide Residues in Human Depot Fat

JOHN R. BROWN, B.Sc., M.D., Ph.D., *Toronto*

Over the past 20 years there has been a considerable increase of new chemicals in our environment, and the incorporation of these materials into human diet is becoming a matter of great concern. The introduction by Müller[1] in 1940 of DDT as an insecticide set the stage for the numerous pesticides which were to follow. These now pose complex health problems, inasmuch as they have become ubiquitous in our biosphere. Of the various groups, the organo-chlorine pesticides are of particular importance because their residue persistence time is greater than that of either the organophosphorus or the carbamate compounds. The recent introduction of electron-capture gas chromatography[2] as an analytical tool has made possible the detection of very small quantities of organo-chlorine pesticides in food products and in human tissues. However, at the present time, it is difficult to assess the biological significance of these minute amounts of pesticide residue, and long-term observations must be carried out before any valid conclusions may be reached.

Pesticides residues in man are built up through ingestion of these materials in food and water, from occupational exposure and from home or garden contamination.

From the Department of Physiological Hygiene, School of Hygiene, University of Toronto, Toronto, Ontario.
Common and chemical names of pesticides mentioned in this text will be found in an appendix following the text.

Reprint requests to: Dr. J. R. Brown, Department of Physiological Hygiene, School of Hygiene, University of Toronto, 150 College St., Toronto 5, Ontario.

Campbell, Richardson and Schafer[3] estimated that approximately 90% of a person's annual intake of DDT-derived material from all environmental sources is ingested with food. Walker, Goette and Batchelor,[4] in a survey of prepared meals, found that "generally those foodstuffs cooked in fat and containing meat, meat combinations, or butter were found to have a higher DDT content than [other] foodstuffs."

The average daily intake of DDT in the samples analyzed was 0.18 mg., and about 34% of the DDT-derived material in the diet was DDE. For an individual of average weight the daily intake of DDT is about 0.0026 mg./kg. body weight. Hayes, Durham and Cueto,[5] during a study on the effect of known repeated oral doses of DDT in man, analyzed ordinary meals for their DDT content and found that there was a considerable variation among foods and even among whole meals with respect to DDT content. The average daily intake of DDT in prepared meals in these samples was found to be 0.202 mg., which was in reasonable agreement with the value of 0.184 mg. reported by Walker, Goette and Batchelor.[4] Hayes *et al.*[6] found that meatless meals served in a cafeteria catering to meat abstainers contained only a quarter as much DDT as did meals served in ordinary restaurants. Analysis of human adipose tissue samples revealed that "persons abstaining from meat deposited in their fat only about one-half the concentration of DDT (2.3 p.p.m.*) as did people in the general population 4.9." They considered that all storage of DDT and DDE in persons not occupationally exposed "results from the presence of these compounds in the diet, especially but not exclusively in fats of animal origin". However, Durham *et al.*[7] found that there was very little DDT or DDE in any of the local Alaskan food they examined, and only small concentrations of DDT (0.8 p.p.m.) and DDE (2.0 p.p.m.) in the body fat of Eskimos eating such food.

The recent work of Durham, Armstrong and Quinby[8] further supports the general conclusion that there is a definite relationship between dietary DDT intake and DDT storage in the body fat. Mills[9] and Williams[10] carried out determinations of the pesticide content of a total diet representing the normal 14-day intake of males, 16 to 19 years of age. The mean DDT and DDE residues were found to be 0.007 and 0.004 p.p.m., respectively.

Organo-chlorine pesticide residues in potable water supplies may be an important source of human contamination with these

*p.p.m. = parts per million. †p.p.t. = parts per trillion.

materials. This pesticide residue in water may be increased through accidental or unauthorized disposal into ground water systems, or from the purposeful application of pesticides to bodies of water or to land during agricultural or other pest control programs. West[11] cites a report by Kraybill describing the presence of lindane at 15-760 p.p.t.† and toxaphene at 10 to 150 p.p.t. in river water and municipal water supplies in Alabama cotton-growing areas. Weibel *et al.*[12] found DDT in a concentration of 1.2 ug./l. in a rainwater sample collected at Cincinnati, Ohio. They also reported the presence of chlordane, heptachlor epoxide, DDT, DDE, Ronnel, dieldrin and 2,4,5-T in a dust cloud covering Cincinnati in January 1965.

Organo-chlorine pesticides generally persist in the soil. Anderson, Deal and Gunther[13] found that the removal of these materials from soil after an application is about 30% per year. Lichtenstein[14] studied the persistence and behaviour of pesticide residues in soil and their translocation into crops. He found residues of 20% of the applied dose of aldrin and heptachlor three years after original application. Harris, Sans and Miles,[15] examining soil residues in the same area, found that DDT and its metabolites were present in amounts in excess of 0.1 p.p.m. in 16 of 31 samples. Aldrin and/or dieldrin were found in amounts in excess of 0.1 p.p.m. in 16 of 31 samples. Wilkinson, Finlayson and Morley[16] found residues of aldrin-dieldrin and heptachlor-heptachlor epoxide in soil nine years after a single treatment. Roebeck *et al.*[17] have studied the effectiveness of water treatment processes in pesticide removal, and concluded that DDT is easily removed by filtration whereas lindane and parathion are not removed by this treatment, and that oxidants such as chlorine and potassium permanganate do not reduce the concentration of the organo-chlorine compounds, but convert parathion into a different but more toxic compound. They also found that fresh, granular, activated carbon beds were able to greatly reduce pesticide concentration to the detectable level of 0.01 ug./l. of water. Human contamination with organochlorine pesticides may occur through occupational exposure to manufacture, formulation or application. Howell[18] found a DDT residue of 17 p.p.m. in the depot fat of a man who had a history of occupational exposure of four years' duration, and who had regularly eaten food known to have an appreciable DDT residue.

It is, therefore, possible that these materials enter the ground water supplies and subsequently contaminate potable sources of

water. A recent survey that I did of ground water in Southern Ontario revealed the presence of lindane, heptachlor epoxide, dieldrin, aldrin and DDE (3.30×10^{-6}, 2.1×10^{-5}, 1.3×10^{-5}, 0.37×10^{-5}, and 1.2×10^{-5} p.p.m., respectively).

Measurable quantities of organo-chlorine pesticides are present in human depot fat, and from a survey of the available data[19] it appears that there has been no appreciable increase in recent years of their DDT and DDE contents. However, there is little information on the concentration of other organo-chlorine pesticides currently present in human depot fat. Specimens of human body fat (gluteal) have been obtained from autopsies carried out in Toronto during 1966, and these have been analyzed for organo-chlorine pesticides.

Materials and Methods

Gluteal-fat samples have been obtained from autopsies of human subjects who died in Toronto from natural or accidental deaths during 1966. As far as is known, none of the subjects had any occupational exposure to organo-chlorine pesticides. Of the 47 samples collected at random, 24 were from men and 23 from women. The mean ages and weights were 61.8 and 63.2 years, and 144.8 and 114.4 pounds for men and women respectively.

Experimental

An F&M Model 400 gas chromatograph equipped with electron capture detector was used. A combination of glass column and on-column injection eliminated loss by decomposition of nanogram quantities of the pesticides investigated.

Instrumental Conditions

Column 6 ft. x 3 mm., ID glass, 4% SE-30 + 6% QF-1 on Chromasorb W, 100/120 mesh.

Temperatures

Column 180° C.
Flash Heater 200° C.
E.C. Detector 200° C.

Carrier Gas Flow Rate

5% Methane–95% Argon. The instrument used was equipped with a pulsed type E.C. detector. For the purpose of the present method the pulse interval was set at 50 microseconds.

Preparation of Column

4% SE-30 + 6% QF-1 on 100/120 mesh acid washed Chromasorb W.SE-30 (0.4 g.) and QF-1 (0.6 g.) were added to a mixture of toluene (85 ml.) and chloroform (10 ml.). The whole was agitated until silicone gum rubber had dissolved and was then left overnight.

Chromasorb W. (10g.) acid-washed 100/120 mesh was added to this mixture and agitated for six hours. The mixture was placed in a round-bottomed flask and the solvent removed under negative pressure in a flash evaporator. When the solvent had been removed, the dry material was transferred to a beaker and stored in an oven at 100° C. for several days.

The columns were packed with this material under negative pressure and conditioned in an oven at 180° C. for 48 hours before use in the gas chromatograph.

It was found that these columns gave a good separation of the chlorinated hydrocarbon pesticides, lindane, heptachlor, DDE, DDT, dieldrin, DDD and aldrin and kelthane.

Procedure

About 200 mg. of adipose tissue was homogenized in an electric homogenizer with 6 ml. acetone (double distilled) for five minutes at 0° C. The contents were transferred to a conical tube containing 35 ml. of acetone which had been previously cooled to –75° to –79° C. by immersion in a mixture of dry ice and methanol. The lipid fraction immediately precipitated. The conical tube was then placed in a mixture of dry ice and methanol and rotated for at least five minutes. The contents were immediately poured into a sintered glass funnel surrounded by a jacket through which methanol at –76° to –79° C. was flowing.

The funnel contained a thin layer of activated charcoal which had been repeatedly washed with pure solvent to remove any interfering substances. The surface of the charcoal was protected with a layer of glass wool which had been similarly treated before use. The precipitated fat was retained in the funnel. The contents of the funnel were then washed with a further 10 ml. of "cold" acetone.

Filtration was carried out under negative pressure. The pressure was adusted to provide a reasonable rate of filtration and yet prevent

the escape of any fat. All filtrates were returned to the "cold bath" before further treatment in order to detect the presence of any residual fat that would interfere with the chromatographic analysis. The filtrate was placed in a flask and the acetone removed on a flash evaporator. The residue was dissolved in 5 ml. of hexane and 4 ul. applied to the gas chromatographic column.

TABLE I.–Mean Per Cent Recovery of Organo-Chlorine Pesticides Added to Human Adipose Tissue Samples

Organo-chlorine pesticides	*Recovery (%)*
Aldrin	93.3
Dieldrin (HEOD)	97.9
Endrin	95.8
Heptachlor	93.9
Heptachlor epoxide	95.3
Kelthane	95.7
Lindane	87.9
p,p′ -DDT	96.1
p.p′ -DDE	94.7
DDD	95.8
Thiodan	91.8

The mean recovery for added organo-chlorine pesticides is given in Table I. The average recovery rate was found to be 93%.

The identities of various pesticides were checked, using the method of thin-layer chromatography with 10-g. samples of fat. For this purpose, layers of silica gel 250 u. thick were prepared on 20 x 20 cm. plates. A thin slurry of silica gel G [Silica Gel G (Stahl), Merck Sharp & Dohme] was prepared using silica gel (30 g.) and silver nitrate (60 ml. 0.5% aqueous solution.) The slurry was immediately spread over five plates with the aid of a variable-thickness applicator. The plates were allowed to stand for five minutes, dried in an oven for 20 minutes at 130° C., cooled and stored in the dark. The plates were used on the day of preparation.

The plates were spotted and were developed with 1% ethyl acetate:hexane until the solvent front had run approximately 10 cm. They were then removed and exposed to ultraviolet radiation until spots appeared.

The addition of silver nitrate to the silica gel plates enhances the sensitivity of the method. These plates also give a good separation of aldrin from DDE. The limits of sensitivity using the method were 0.1 ug. for p,p′ -DDT and 0.5 ug. for DDE, HEOD, lindane, aldrin and heptachlor epoxide.

Results

The mean (arithmetic) concentrations of organo-chlorine pesticide in 47 samples of human adipose tissue are given in Table II. Dieldrin was not present in 12, lindane in five, aldrin in seven, DDD in 20, or heptachlor epoxide in 17 of the 47 fat samples analyzed for their organo-chlorine content.

TABLE II,–Organo-Chlorine Pesticides in Human Adipose Tissue (Toronto, 1966)

Organo-chlorine pesticide	*No. of samples*	*Concentration (p.p.m.)*		
		Mean	*SD*	*Range*
p,p′ -DDT	47	1.09	.49	.28 - 2.65
p,p′ -DDE	47	2.66	1.32	.6 - 6.8
HEOD	35	.22	.12	.07 - .53
Lindane	42	.07	.04	.01 - .18
Aldrin	40	.03	.02	.01 - .14
DDD	27	.30	.19	.01 - .90
Heptachlor epoxide	22	.14	.09	.01 - .40

The concentrations of DDE and DDD may be expressed in terms of derived DDT by multiplying the p.p.m. for these substances by factors of 1.1148 and 1.1076, respectively. Using these factors, the mean value for DDT plus DDT-derived material has been calculated to be 4.39 p.p.m.

The mean concentrations of DDT and DDE were 1.09± .49 and 2.66± 1.32 p.p.m., respectively, and the average ratio of DDE:DDT was 2.44.

Table III gives the mean (arithmetic) concentrations of organo-chlorine pesticides in human adipose tissue in relation to the sex of the subject. The mean concentrations for men are higher than those for women. However, these differences are not statistically significant. The average ratio of DDE:DDT was 2.61 and 2.37 for men and women, respectively.

TABLE III.–Organo-Chlorine Pesticides in Human Adipose Tissue in Relation to Sex of Subject

Organo-chlorine pesticide	*Sex*	*No. of samples*	*Mean weight (lbs.)*	*Mean age (years)*	*Organo-chlorine pesticide concentration (p.p.m.)* Mean	SD	Range
p,p′ -DDT	M	24	141.6	62.1	1.16	.43	.28 - 2.03
	F	23	114.2	65.2	0.98	.33	.40 - 2.65
p,p′ -DDE	M	24	141.6	62.1	3.03	1.28	1.2 - 6.8
	F	23	114.2	65.2	2.32	1.18	0.6 - 5.9
Lindane	M	21	142.1	61.6	0.08	.044	.01 - .18
	F	21	112.9	66.0	0.07	.040	.02 - .18
HEOD	M	20	147.4	64.4	0.23	.09	.08 - .39
	F	15	118.4	64.4	0.20	.14	.07 - .53
Aldrin	M	20	140.4	63.4	0.042	.034	.01 - 0.14
	F	20	112.7	61.2	0.028	.015	.01 - 0.07
DDD	M	15	146.8	63.7	0.34	.18	.01 - 0.90
	F	12	112.9	64.4	0.27	.12	.01 - 0.44
Heptachlor epoxide	M	22	144.6	65.8	0.15	.31	.01 - 0.40
	F	8	111.4	67.1	0.11	.16	.02 - 0.24

TABLE IV.–Average Concentration (p.p.m.) of Organo-Chlorine Pesticides in Human Adipose Tissue in Relation to Age

Age Groups	*<40*	*>40*
Pesticide		
p,p′ -DDT	.75	1.18
p.p′ -DDE	1.46	2.84
DDD	.28	.30
Aldrin	.03	.03
HEOD	.16	.24
Heptachlor epoxide	.08	.14
Lindane	.06	.08
Total pesticide	2.82	4.81

The concentrations of organo-chlorine pesticides in human adipose tissue have been examined in relation to age (Table IV). In general, adipose tissue samples derived from subjects less than 40 years of age have somewhat lower organo-chlorine pesticide residues than those obtained from older subjects. The ratio of organo-chlorine pesticide for those under 40 to those over 40 is 1:1.71. As well as an absolute increase in p,p′ -DDT and p,p′ -DDE with age, there is also an increase in the ratio of DDE/DDT, namely 1.94 to 2.41.

Discussion

A similar study was carried out by Hoffman, Fishbein and Andelman[21] on fat samples obtained from 282 autopsies. These results have been compared with those obtained in the present study. Although their mean values for p,p′ -DDT, p,p′ -DDE and lindane were higher (2.9, 7.4 and 0.57 p.p.m., respectively) than those for the present samples, only that for p,p′ -DDT is significantly higher ($P < .05, > .02$).

They found that the DDE, on an average, constituted 72% of the total DDT-derived material. In the present samples the average was 72.6%.

Read and McKinley[22] analyzed 62 fat samples obtained in Vancouver, Winnipeg and Toronto (1959-1960). They found that the average DDT plus DDE was 4.9 p.p.m.; on the average, 67% of this was present as DDE. This compares well with the present finding of 3.75 p.p.m.

Hayes, Dale and LeBreton[23]have reported a mean lindane residue of 1.19 p.p.m. in 10 human fat samples of French origin. Hunter,

Robinson and Richardson[24] report a mean residual of 0.21 p.p.m. HEOD in fat samples obtained from Southern England. This is comparable to the mean values of 0.22 p.p.m. obtained for the present Canadian samples. Robinson *et al.*[19] were unable to detect endrin or heptachlor expoxide in 91 and 20 samples, respectively. However, they reported 0.015 p.p.m. as the mean residual concentration of lindane in their particular human fat samples.

The mean residual concentrations of organo-chlorine pesticides in human adipose tissue samples, obtained at different times and from different locations, have been summarized in Table V. It is seen that the present Canadian estimate is in agreement with that of Read and McKinley[22] in 1959-1960, and with the United Kingdom estimate. In the Indian estimates[32] there is a reversal of DDE/DDT ratio. This is thought to be due to the fact that most Indians eat little meat and would, therefore, be receiving a relatively low proportion of DDE in their diet. Animal metabolism of DDT tends to increase the proportion of DDE above that originally ingested.

At the present time the organo-chlorine pesticide residue of human adipose tissue samples for the general population is lower than that for the general population of the United States[31] and of Israel.[30]

In general it would appear that the earlier determinations of total DDT carried out by the Schechter-Haller method[33] are in some instances higher than determinations by gas chromatographs using either the micro-coulometric or electron-capture detector. Dale and Quinby[28] compared the results of adipose tissue analysis for organo-chlorine pesticides using microcoulometric gas chromatography and colorimetric analysis (Schechter-Haller). They found that the total DDT residues obtained by means of colorimetric analysis were, on an average, 1.6 times greater than those obtained by gas chromatography.

New environmental health problems are created by chemicals including pesticides. Acute exposure, however, is not the major problem, but rather the problem is the delayed effects of the accumulation of small amounts of chemical contaminant within the body tissues.

The significance of the present concentration of organo-chlorine pesticide residues in human adipose tissue is at present little understood.

TABLE V.–Organo-Chlorine Pesticide Residues in Human Adipose Tissue

Date	Country of origin	No. in sample	Organo-chlorine pesticide residue (p.p.m.) p,p'-DDT	p,p'-DDE	DDT+DDE	Lindane	HEOD	Aldrin	Heptachlor epoxide	References
1942x	U.S.A	10	–	–	–	–	–	–	–	5
1950x	U.S.A.	75	5.3	–	–	–	–	–	–	25
1954-1956x	U.S.A.	61	4.9	6.2	11.1	–	–	–	–	5
1959-1960x	Canada	62	1.6	3.3	4.9	–	–	–	–	22
1960x	Alaska	20	0.8	2.2	3.0	–	–	–	–	7
1958-1959x	Germany	60	1.0	1.2	2.2	–	–	–	–	26
1960x	Hungary	50	5.7	6.7	12.4	–	–	–	–	27
1961x	France	10	1.7	3.5	5.2	1.19	–	–	–	23
1961-1962xxx	U.K.	131	–	–	2.2*	–	0.21*	–	–	24
1961-1962xx	U.S.A.	30	1.14	3.82	4.96	0.20	0.15*	–	–	28
1961-1962xx	U.S.A.	130	4.0	7.8†	11.8	–	–	–	–	49
1962-1963xx	U.S.A.	282	2.9	7.4	10.3	–	–	–	–	
		64	–	–	–	–	0.11	–	–	21
1964xxx	U.K.	100	1.0*	2.3*	3.3*	–	–	–	–	
		20	–	–	–	–	0.21*	–	–	19
N.S.										
1964	U.S.A.	64	2.35	4.63	6.98	–	0.31	–	0.10	31
1963-1964xxx	U.K.	65	1.1*	2.0*	3.3	–	0.26	–	.10	29
1963-1964x	Israel	8.5	10.7	19.2	–	–	–	–	–	30
1964xx	India (Delhi)	24	13.5	11.6	25.1	0.28	0.03	–	–	32
1964xx	U.S.A.	25	1.7	6.9	8.6	–	0.29	–	–	48
1966xxx	Canada	27	1.1	2.7	3.8	0.07	0.22	.03	0.14	This communication

Method of estimation:
x–Schechter-Haller
xx–Gas chromatograph and microcoulometric detection.
xxx–Gas chromatography and electron capture detection.

*–Geometric mean.
†–Based on 121 samples.

Hayes, Durham and Cueto[5] fed two groups of human volunteers with 3.5 and 25 mg. DDT respectively, for periods ranging up to 18 months; the second dose was about 200 times greater than the amount the average non-exposed, non-vegetarian individual takes each day in his diet. They noted that "During the entire study, no volunteer complained of any symptoms or showed, by the tests used, any sign of illness that did not have an easily recognized cause, clearly unrelated to exposure to DDT." The highest DDT fat residue found during the study was 340 p.p.m. Ortelee[34] carried out clinical examinations on a group of DDT formulating plant workers, 65% of whom had been exposed to an equivalent oral dose of 35 mg. DDT per day over a period of three to five years. He detected no ill effects in these workers, as judged by clinical examination or their work records, and concluded that, with the possible exception of a rare hypersensitivity reaction, it was unlikely that exposure to current levels of DDT contamination would produce chronic DDT poisoning in the general population. Hayes[35] points out that organo-chlorine pesticides are cumulative in human adipose tissue, their pharmacological action being largely in the central nervous system, causing hyperexcitation, generalized tremors, spastic or flaccid paralysis and convulsions. He states that DDT administered to a man at a rate of 0.2 g. mg./ kg./ day will cause a mild illness and 0.5 g./kg./ day may be fatal.

That there has been no appreciable increase in the residue DDT concentration in human adipose tissue over recent years indicates that the residue of this agent in the diet has reached a state of equilibrium. Hayes, Durham and Cueto[5] showed that the storage of DDT in man is proportional to dosage and that storage reaches equilibrium in about a year despite continued intake.

Over a period of up to nine years, Hoogendam, Versteeg and de Vlieger[36] studied over 300 shiftworkers employed in the manufacture of aldrin, dieldrin and endrin. Over 1300 man-years' exposure to these organo-chlorine pesticides in a concentrated form did not cause any fatalities or any permanent injury. The workers were under thorough medical supervision, including periodic electroencephalographic examination and liver function tests. Seventeen of the workers had abnormal electroencephalograph rhythms, but recovered completely when removed from exposure. No impairment of liver function was detected.

Falk, Thompson and Kotin[37] reported that, following periods of exposure to high dietary levels of organo-chlorine pesticides, an

increased incidence of benign and malignant tumours of the liver has been observed in rodents and certain fish. Fitzhugh and Nelson[38] found that commercial DDT produced hepatomas in rodents at levels of 200 to 800 p.p.m. in near-life-span exposure. Davis and Fitzhugh[39] showed that aldrin and dieldrin caused tumours in mice when added to the diet at a level of 10 p.p.m.

In a study of apple growers, Davignon *et al.*[40] found a greater incidence of neurological manifestations and leukopenia among them than among the general population. Loge[41] described the sporadic occurrence of a myelogenic blood dyscrasia following exposure to benzene hexachloride. Lindane has been implicated as a cause of blood dyscrasia: the American Medical Association's Council on Drugs Registry[42] on blood dyscrasias lists 18 reports of major blood dyscrasias in which lindane was implicated. Additional cases have been reported by Marchand, Dubrulle and Goudemand,[43] Albarhary and Dulrisay,[44] Scott, Cartwright and Wintrobe,[45] Sanchez-Medal, Castanedo and Garcia-Rojas,[46] and Mastromatteo.[47]

The ready availability of formulations that may contain one or more organo-chlorine pesticides is a potential source of human contamination. Until recently, shelf paper impregnated with lindane was available for use in kitchen food-storage areas. Dispensers which intermittently or continually release these materials into the air can be purchased for use in homes, schools, restaurants and places of work. Pesticides are available in a great variety of sprays, dusts and pellets, for use in home and garden or on pets. Much is known concerning the effect of environmental contamination of wild life, but the possible significance of exposure to pesticides in the home and garden has been largely unexplored.

At the present time there is little evidence to suggest that the current levels of deposition of organo-chlorine pesticides in human tissue presents any threat to the health of the general community. Any untoward increases should, however, be viewed with suspicion.

Summary

Organo-chlorine pesticide residues were determined in 47 human adipose tissue samples. The concentration of these materials in Canadian samples does not appear to have increased since 1959-1960. The biological significance of pesticide residues in human adipose tissue is discussed. At the present time these do not appear to constitute a serious threat to health.

Résumé

L'auteur étudie les résidus des insecticides organo-chlorés dans des échantillons de tissu adipeux chez 47 sujets. La concentration de cette substance chez les sujets canadiens ne semble pas avoir augmenté depuis 1959-1960. Il discute l'importance biologique des résidus des insecticides dans le tissu adipeux chez l'homme. A l'heure actuelle, il ne semble pas en résulter de menace sérieuse à la santé nationale.

REFERENCES

1. MULLER, P.: cited by Tegeris, A. S. *et al.:* Arch. Environ, Health (Chicago), **13**: 787, 1966.
2. LOVELOCK, J. E. and LIPSKY, S. R. : *J. Amer. Chem. Soc.,* **92**: 431, 1960
3. CAMPBELL, J. F., Richardson, L. A. and SCHAFER, M. L.: *Arch. Environ. Health (Chicago),* **10**: 831, 1965.
4. WALKER, K. C., GOETTE, M. B. and BATCHELOR, G. S.: *Journal of Agricultural and Food Chemistry,* **2**: 155, 1954.
5. HAYES, W. J., Jr., DURHAM, W. F. and CUETO, C.: *J. A. M. A.,* **162**: 890, 1956.
6. HAYES, W. J., Jr. *et al.: A.M.A. Arch. Industr. Health.* **18**: 398, 1958.
7. DURHAM, W. F. *et al.: Science.* **134**: 1880, 1961.
8. DURHAM, W. F., ARMSTRONG, J. F. and QUINBY, G. E. *Arch. Environ. Health (Chicago),* **11**: 641, 1965.
9. MILLS, P. A.: *J. Ass. Agric. Chem.,* **46**: 742, 1963.
10. WILLIAMS, S.: *Ibid.,* **47**: 815, 1964.
11. WEST, I.: *Arch. Environ. Health (Chicago),* **9**: 626, 1964.
12. WEIBEL, S. R. *et al.: Journal American Water Works Ass.,* **58**: 1075, 1966.
13. ANDERSON, L. D., DEAL, A. S. and GUNTHER, F. A.: Insecticide residues in soil and their effects on vegetable crops. Cited by West, I.: *Arch. Environ. Health (Chicago),* **9**: 626, 1964.
14. LICHTENSTEIN, E. P.: *Arch. Environ. Health (Chicago),* **10**: 825, 1965.
15. HARRIS, C. R., SANS, W. W. and MILES, J. R. W.: *Journal of Agriculture and Food Chemistry,* **14**: 398, 1966.
16. WILKINSON, A. T. S., FINLAYSON, D. G. and MORLEY, H. V.: *Science,* **143**: 681, 1964.
17. ROEBECK, G. G. *et al.: Journal American Water Works Ass.,* **57**: 181, 1965.
18. HOWELL, D. E.: *Proceedings of the Oklahoma Academy of Science,* **29**: 31, 1948.
19. ROBINSON, J. *et al.: Brit. J. Industr. Med.,* **22**: 220, 1965.
20. McKINLEY, W. P., SAVARY, G. and WEBSTER, C.: *Journal of Agricultural and Food Chemistry,* **10**: 226, 1962.
21. HOFFMAN, W. S., FISHBEIN, W. I. and ANDELMAN, M. B.: *Arch. Environ, Health (Chicago),* **9**: 387, 1964.
22. READ, S. T. and McKINLEY, W. P.: *Ibid.,* **3**: 209, 1961.
23. HAYES, W. J., Jr., DALE, W. E. and LE BRETON, R.: *Nature (London),* **199**: 1189, 1963.
24. HUNTER, C. G., ROBINSON, J. and RICHARDSON, A.: *Brit. Med. J.,* **1**: 221, 1963.

26. MAIER-BODE, H.: *Med. Exp. (Basel)*, **1**: 146, 1959.
27. DENES, A.: *Nahrung*, **6**: 48, 1962.
28. DALE, W. E. and QUINBY, G. E.: *Science*, **142**: 593, 1963.
29. EGAN, H. *et al.: Brit. Med. J.*, **2**: 66, 1965.
30. WASSERMAN, M. *et al.: Arch. Environ. Health (Chicago)*, **11**: 375, 1965.
31. ZAVON, M. R., HINE, C. H. and PARKER, K. D.: *J. A. M. A.*, **193**: 837, 1965.
32. DALE, W. E., COPELAND, M. F. and HAYES, W. J., Jr.: *Bull, W.H.O.*, **33**: 471, 1965.
33. SCHECHTER, M. S. *et al.: Industrial and Engineering Chemistry, Anal. ed.*, **17**: 704, 1945.
34. ORTELEE, M. F.: *A.M.A. Arch. Industr. Health*, **18**: 433, 1958.
35. HAYES, W. J., Jr.: *Amer. J. Public Health*, **45**: 478, 1955.
36. HOOGENDAM, I., VERSTEEG, J. P. J. and DE VLIEGER, M.: *Arch. Environ. Health (Chicago)*, **10**: 441, 1965.
37. FALK, H. L., THOMPSON, S. J. and KOTIN, P.: *Ibid.*, **10**: 847, 1965.
38. FITZHUGH, O. G. and NELSON, A. A.: *J. Pharmacol. Exp. Ther.*, **89**: 18, 1947.
39. DAVIS, K. J. and FITZHUGH, O. G.: *Toxic. App. Pharmacol.*, **4**: 187, 1962.
40. DAVIGNON, L. F. *et al.: Canad. Med. Ass. J.*, **92**: 597, 1965.
41. LOGE, J. P.: *J. A. M. A.*, **193**: 110, 1965.
42. BEST, W. R.: *Ibid.*, **185**: 286, 1963.
43. MARCHAND, M., DUBRULLE, P. and GOUDEMAND, M.: *Arch. Mal. Prof.*, **17**: 256, 1956: Abstract, *Bull. Hyg. (Lond.)*, **31**: 1023, 1956.
44. ALBARHARY, C. and DULRISAY, J.: *Ibid.*, **18**: 582, 1957.
45. SCOTT, J. L. CARTWRIGHT, G. E. and WINTROBE, M. M.: *Medicine (Balt.)*, **38**: 119, 1959.
46. SANCHEZ-MEDAL, L., CASTANEDO, J. P. and GARCIA-ROJAS, F.: *New Eng. J. Med.*, **269**: 1365, 1965.
47. MASTROMATTEO, E.: *Canad. Med. Ass. J.*, **90**: 1166, 1964.
48. HAYES, W. J., Jr., DALE, W. E. and BURSE, V. W.: *Life Sci.*, **4**: 1611, 1965.
49. QUINBY, G. E. *et al.: J. A. M. A.*, **191**: 175, 1965.

APPENDIX:

Common and Chemical Names of Pesticides Mentioned in the Text

ALDRIN. 1,2,3,4,10,10-hexachloro-1,4,4a,5,8,8a-hexahydro-1,4-endo,exo,5,8-dimethanonaphthalene
CHLORDANE. 2,3,4,5,6,7,8,8-octachloro,2,3,3a,4,7,7a-hexahydro-4,7-methanoindene
DDT (p,p′-isomer). . . . . .2,2-bis (p-chlorophenyl)1,1,1-trichloroethane
DDE (p,p′-isomer). . . . . .2,2-bis (p-chlorophenyl)-1,1-dichloroethylene
DDD. . . . . 2,2 bis (p-chlorophenyl)-1,1-dichloroethane
DIELDRIN. . . . . 1,2,3,4,10,10 -hexachloro-exo-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,-endo,exo-5,8-dimethanonaphthalene
ENDRIN. 1,2,3,4,10,10-hexachloro-exo-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4,5,8-endo,endo-dimethanonaphthalene
HEPTACHLOR. 1,4,5,6,7,8,8-heptachloro-3a,4,5,5a-tetrahydro-4,7-endomethanoindene

HEPTACHLOR EPOXIDE	1,4,5,6,7,8,8-heptachloro-2,3-epoxy-2,3,3a,7a-tetrahydro-4,7-methanoindene
LINDANE γ-ISOMER	1,2,3,4,5,6-hexachlorocyclohexane
RONNEL	0,0-dimethyl-0-2,4,5-trichlorophenyl phosphorothioate
2,4,5-T	2,4,5-trichlorophenoxyacetic acid
BENZENE HEXACHLORIDE	1,2,3,4,5,6-hexachloro-cyclohexane, mixed isomers
TOXAPHENE	chlorinated camphene containing 67-69% chlorine
THIODAN	6,7,8,9,10,10-hexachloro-1,5,5a,6,9,9a-hexahydro-6,9-methano-2,3,4-benzodioxathiepin3-oxide
KELTHANE	4,4'-dichloro-α-(trichloromethyl) benzhydrol
PARATHION	0,0-diethyl 0-p-nitrophenyl phosphorothioate

APPENDIX II

Toxicological Aspects of CHCP

Wayland J. Hayes, Jr., M.D., Ph.D.
Professor of Biochemistry
Vanderbilt University School of Medicine
Nashville, Tennessee

Although this paper is a mere outline of the toxicology of chlorinated hydrocarbon insecticides, it attempts to emphasize certain aspects that are sometimes neglected and to recall criteria for judging the net value of these compounds. The paper is written from the standpoint of human safety but certain variables are mentioned that may influence the safety of pesticides to other species.

PHARMACOLOGY

Absorption

All chlorinated hydrocarbon insecticides may be absorbed by the skin as well as by the lungs and gastrointestinal tract. The efficiency of skin absorption is low for lindane, toxaphene, and especially DDT but is high enough for aldrin, chlordane, dieldrin, endrin, heptachlor, isodrin, and endosulfan that these compounds are only two to three times more toxic by mouth than when applied to the skin.

Metabolism

The metabolism of the chlorinated hydrocarbon insecticides has been reviewed (Hayes, 1965). Apparently all these compounds are converted by mammals to more water-soluble materials and excreted in that form both in the urine and in the feces. The metabolism of DDT has been worked out in considerable detail and that of aldrin and dieldrin is reasonably well known. Details of the metabolism of the other chlorinated hydrocarbon insecticides are understood poorly.

Judging from the relation of storage to dosage, the rate of metabolism decreases in approximately the following order: methoxychlor > endrin > aldrin and dieldrin > DDT > mirex. Thus, little or no methoxychlor is stored in tissues even when dosage is substantial.

Distribution and Storage

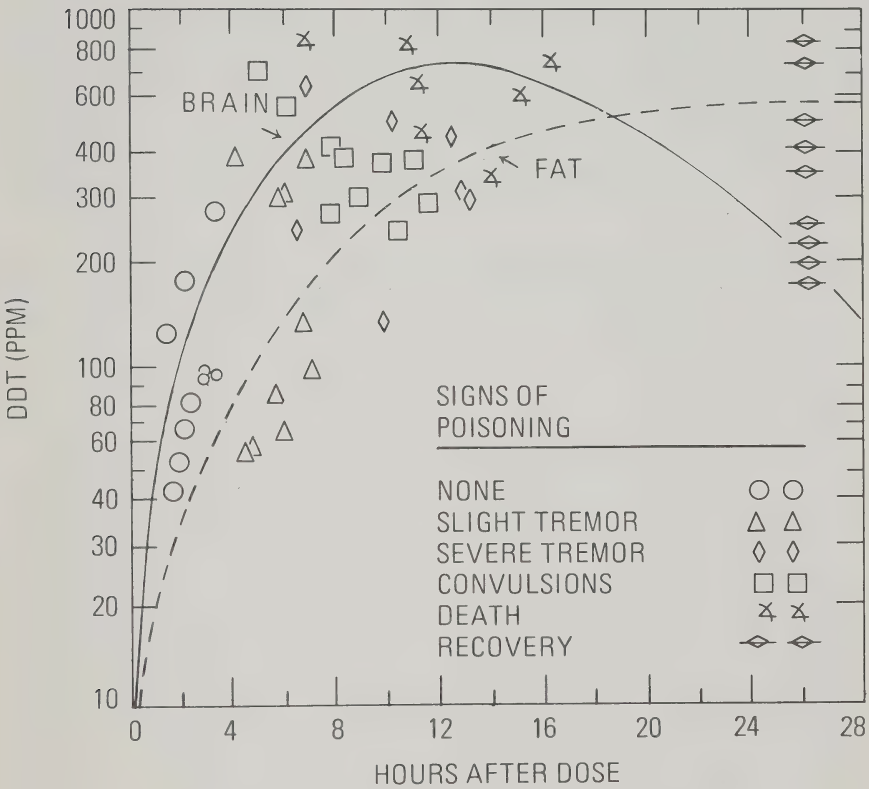
All the chlorinated hydrocarbon insecticides are highly soluble in fat and they are stored preferentially in adipose tissue. However, they are stored to some extent in all tissues. In general, the concentration in the viscera, including the brain, is more similar to the concentration in blood than it is to the concentration in fat when storage has reached a steady state. Different authors have found the equilibrium concentration of DDT in human fat to be 306 to 344 times greater than that in plasma (Robinson and Hunter, 1966; Laws *et al.*, 1967). The equilibrium concentration of dieldrin in fat is 136 to 247 times greater than its concentration in plasma (Hunter *et al.*, 1969; Hayes and Curley, 1968). However, soon after a single dose, the concentration of one of these compounds in the brain may be as great or greater than that in the blood (Dale *et al.*, 1963).

For any given compound and species, the degree of illness is proportional to the concentration of insecticide in the nervous system as measured in brain samples (Dale *et al.*, 1963). This is true, whether dosage is acute, subacute or chronic (Hayes and Dale, 1964).

TABLE 1 – Concentration (ppm) of DDT in the brain and fat of rats. The animals receiving 1 to 9 doses were subject to no other stress. The rats receiving 90 doses at 10 mg/kg/day were put on half rations from the 91st to 101st day of dosing. See Hayes and Dale, 1964.

No. Doses	Condition	Tissue	Pre-stress	Stress		Re-covery
				Died	Lived	
1	150 mg/kg	Brain	<1	737	404	176
		Fat	8	361	213	598
9	60 – 120 mg/kg/day	Brain	<1	600	589	268
		Fat	8	6,660	2,484	5,359
90	10 mg/kg/day plus Starvation at half dosage	Brain	92	595	166	287
		Fat	621	5,574	988	593

FIGURE 1 – Concentration of DDT in the lipids of brain (solid symbols) and fat (open symbols) of rats at different times after a single dose of the compound at the rate of 150 mg/kg. Different symbols indicate the clinical state of each animal when the samples were taken. After Dale *et al.* (1963).

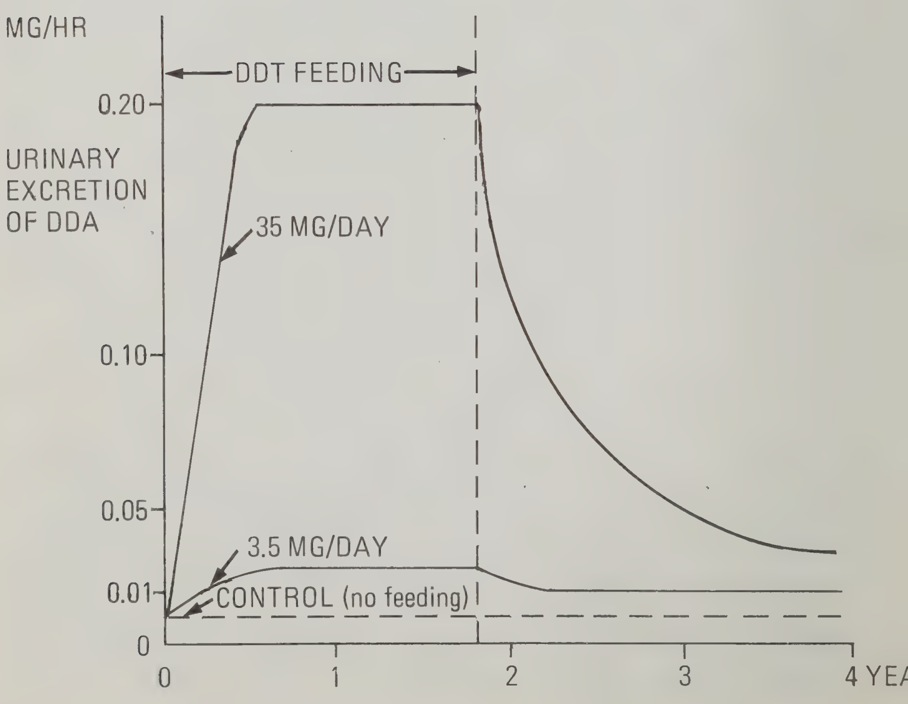


An illustration of these relationships is shown in Figure 1 and Table 1. Unlike the situation in the brain, the concentration of insecticide in body fat of animals killed by DDT varies greatly depending on whether dosage was acute or chronic. Thus, if analytical values are to be used in diagnosing the cause of death, the samples must be taken from the nervous system or there must be information on the duration of exposure leading to death.

It appears that the concentration of a particular chlorinated hydrocarbon insecticide in the brain necessary to produce death is of the same order of magnitude in different warm-blooded animals (Hayes, 1965). However, this generalization must be accepted with caution because only a few compounds and species have been studied in this regard.

When repeated doses of any compound are given in such a way that a part of the material still remains in the body when the second dose is absorbed, the concentration in the blood and tissues after the second dose will be greater than that after the first. This increase in storage continues at a gradually decreasing rate until a steady state is reached when the amount lost from the body each day is equivalent to the amount absorbed. Just how rapidly this steady state will be

FIGURE 2 – Excretion of DDA (mg/hr) by volunteers fed DDT at rates of 0, 3.5 and 35 mg/man/day. Unpublished data.



reached depends on the rate of metabolism and excretion. The reason that an equilibrium occurs at all is that the amount excreted is proportional, not to the dose, but to the concentration of material in the blood. When the rate of loss is rapid, equilibrium will be reached quickly and its level will be relatively low. When excretion is inefficient, it takes a long time to reach equilibrium and storage is high relative to the daily dosage. A steady state of storage of many drugs is reached within a few days. The chlorinated insecticides reach a steady state of excretion and storage in a matter of months or years, depending on the compound and the species (see Figure 2). Certain of the heavy metals reach a steady state of storage only after decades if, in fact, it is ever achieved in a life-time. Thus, although the chlorinated hydrocarbon insecticides are not stored nearly so avidly as are the heavy metals, they are stored much more tenaciously than most drugs and, in fact, most other insecticides.

It was shown very early (Fitzhugh and Nelson, 1947) that some rats that had received large doses of DDT became poisoned when they were starved. A quantitative study by Dale *et al.* (1962) confirmed this finding but showed that biotransformation of DDT and the excretion of metabolites increased when the compound was mobilized along with fat. The rats that were able to keep their blood – and, therefore, brain – levels low enough remained well; the others became sick. Doubling of the rate of excretion of dieldrin in the face of starvation has been demonstrated (Heath and Vandekar, 1964). In fact, excretion of dieldrin by rats is so efficient that it has been impossible to precipitate dieldrin poisoning in this species by starvation. It was pointed out by Dale *et al.* (1962) that human poisoning associated with starvation is unlikely because people simply do not store enough insecticide and do not mobilize their fat rapidly enough. The situation may be entirely different for some species. Metabolic rate tends to be inversely proportional to body weight. Thus, small anumals are more likely to be in danger than large ones. However, the final outcome depends on the rate at which the mobilized insecticide is metabolized and excreted. Thus, each compound must be judged separately in each species.

Excretion

Only by using radioactively tagged compounds has it been possible to account by excretion for all the insecticide lost from the body. Chemical methods fail to account for all of the excreted material, partly because some of the metabolites can not be extracted from the excreta and partly because some of the

metabolites are not detected by available analytical methods. In spite of these defects of chemical analysis, one or more metabolites of the important chlorinated hydrocarbon insecticides can be measured in urine and may be used for monitoring workers or even people in the general population.

It is interesting that the proportion of different metabolites of an insecticide often vary depending on the dosage rate. The difference is probably related to the stimulation of the microsomal enzymes of the liver by higher doses, and similar changes can be produced by using other enzyme inducers.

Mode of Action and Cause of Death

The mode of action of the chlorinated hydrocarbon insecticides is unknown, but obviously it involves the nervous system. Limited studies have emphasized the importance of the action of a compound on a particular part of the brain. However, action on all parts of the nervous system has been found when looked for properly. It is probably the sum of action rather than any one segment of it that is important.

Although these insecticides may affect other systems and organs, notably the liver, these actions are distinctly secondary. Several chlorinated hydrocarbon insecticides have been shown to inhibit one or more enzymes and their actions on drug-metabolizing enzymes is an important source of their side effects. However, there is no convincing evidence that any of these inhibitions is related to the basic mode of action of the compound and the cause of death. In fact, the possibility has not been excluded that these compounds act physically just as many organic vapors are thought to do on the basis of their thermodynamic activities or chemical potentials. These values are in about the range of 0.01 to 0.1 for anesthetic or other drug action and in the range of 0.1 to 1.0 for the lethal action of compounds that act physically (Ferguson, 1939). Compounds that act chemically have values of about 0.001 even for lethality, and lower for drug effects. The difficulty is that the vapor pressures and solubilities of the chlorinated hydrocarbon insecticides are so low that their chemical potentials are difficult to estimate.

Side Effects

Two important kinds of side effects of some chlorinated hydrocarbon insecticides involve their direct estrogenic action

(Kupfer, 1967) and their ability to induce microsomal enzymes of the liver (Durham, 1967; Conney, 1967).

The estrogenic action of DDT was known at least as early as 1950 (Burlington and Lindeman, 1950). It does not parallel toxicity, and some analogs and metabolites are relatively active. Doses that have been effective in any mammal are larger than doses received by the most heavily exposed workers, much less the general public. The same assurance extends to domestic animals including birds. However, the estrogenic action frequently is more marked in birds than in mammals, and might have practical implications for some wild species.

The stimulatory effects of foreign compounds on liver microsomal enzyme activity was discovered independently by Brown *et al.* (1954) and by Remmer *et al.* (1958a and 1958b). Since that time it has been found that this activity is stimulated by several chlorinated hydrocarbon insecticides and their analogs, including DDT, DDE, DDD, perthane, methoxychlor, BHC, chlordane, aldrin, dieldrin, endrin heptachlor and heptachlor epoxide. It is clearly established that the liver microsomal enzymes metabolize androgens, estrogens, and various adrenal steroids as well as foreign compounds. Sometimes this property can be put to use as in the treatment of adrenal hypertrophy with massive doses of o,p′ -DDD. However, the highest doses absorbed by formulators and applicators of chlorinated hydrocarbon insecticides have no detectable clinical effect. Studies to determine whether there are subclinical effects on these enzymes in the most heavily exposed workers are planned.

Reversibility

Animals that survive poisoning by a chlorinated hydrocarbon insecticide recover completely. The relatively minor morphological changes produced by the compounds are reversible. Of course, clinical and morphological effects may continue as long as the concentration of toxicant is sufficiently high. Although the rate of metabolism and excretion of some of the compounds is slow, it is not sufficiently slow to pose a practical problem as in the case of mercury and lead. Actually, most human cases of poisoning by these compounds have followed a single large dose. The prompt recovery of patients who survived the first few hours is readily explained by rapid redistribution of toxicant to the muscles and somewhat later to the fat (Garrettson and Curley, in press) and is not seriously complicated by the fact that total elimination of the dose is slow.

EFFECT OF A SINGLE DOSE IN ANIMALS

Symptomatology

All of the insecticidally active isomers of chlorinated hydrocarbon insecticides are stimulants of the nervous system, and large doses of them lead to convulsions. Some of the compounds, notably DDT, produced tremor, a condition closely related to normal shivering and apparently the result of direct action on the part of the brain that controls shivering. Large doses of several of the compounds (and perhaps all of them) can interfere centrally with temperature control and thus lead to fever. Some compounds, notably DDT, sensitize the myocardium to injected epinephrin or even the amount of epinephrin released during a seizure. Thus, in some species, ventricular fibrillation may be the cause of death, but in others the fibrillating heart usually returns to normal rhythm.

Irritation and Sensitization

Chlorinated hydrocarbon insecticides cause little irritation or sensitization. It is true that a few outbreaks of sensitization have been reported in connection with benzene hexachloride, but they occurred during relatively brief periods in limited areas; it seems likely that they were caused by some by-product of manufacturing or by some unusual ingredient in the formulation.

Toxicity

Table 2 shows the oral LD_{50} for a number of chlorinated hydrocarbon insecticides. As may be seen, isodrin is about 400 times more poisonous than methoxychlor. The toxicity of the other compounds lies somewhere between these two. None of the chlorinated hydrocarbon insecticides is as poisonous as several of the organic phosphorus insecticides in common use.

Pathology

A fatal dose of any chlorinated hydrocarbon insecticide produces so little pathology that it entirely fails to explain death. Furthermore, there is nothing characteristic about this pathology. A diagnosis can not be made on the basis of pathology alone.

EFFECT OF REPEATED DOSES IN ANIMALS

Symptomatology

The signs of illness produced by repeated doses of each chlorinated hydrocarbon insecticide are entirely similar to the signs

TABLE 2 – Toxicity of chlorinated hydrocarbon insecticides and certain of their metabolites to rats. All values are for male rats as given by Gaines (1969) except as indicated.

	1-Dose LD_{50} (mg/kg)	90-Dose LD_{50} (mg/kg)	Chronicity Factor
Aldrin	39		
Chlordane	335		
Chlorbenzilate	1040		
DDA	740		
DDD	4000		
DDE	880		
DDT (Tech)	250[a]	46[a]	5.4[a]
p,p′-DDT	113		
Dicofol	1100		
Dieldrin	102	8.2[a]	12.8[a]
Dilan	600		
Endosulfan	43		
Endrin	18		
Heptachlor	100		
Heptachlor epox.	34[c]		
Isodrin	15		
Kepone	125		
Lindane	88		
Methoxychlor	6000[d]		
Mirex	365[b,e]	6.0[b,e]	60.8[b,e]
Perthane	4000		
Toxaphene	90		

a/ Unpublished data of T. B. Gaines.
b/ Test in female rats.
c/ Velsicol Corp.
d/ Lehman (1951).
e/ Gaines and Kimbrough, 1969.

produced by a somewhat larger single dose. Signs of illness may continue with or without interruption in response to continuing dosage. If dosage is stopped and the animal survives, recovery may take slightly longer than it does after a single dose. The small difference is conditioned by the presence of marked storage that always accompanies repeated doses sufficiently large to lead to

poisoning. As discussed in the section on distribution and storage, even the mobilization of toxicant along with fat during starvation has not been a source of illness in large animals. Whether such mobilization will constitute a problem in small animals depends both on the species and the particular compound involved.

Toxicity

It is only recently that any effort has been made to measure the effect of repeated doses of compounds and express the result numerically as is almost universally done in connection with acute toxicity. It has been shown both in rats and dogs and in connection with a wide range of compounds that the effects measured in 90 days are similar to those measured following a lifetime of dosage (Weil and McCollister, 1963). Therefore, it has been suggested that the effect of repeated doses be expressed in the form of a 90-dose LD_{50} It also has been suggested that the ratio between the 1-dose LD_{50} and the 90-dose LD_{50} be expressed as a quotient to be called the *chronicity factor* and used as a measure of the ability of the compound to produce cumulative effects (Hayes, 1967b). Table 2 gives 90-dose LD_{50} values and the chronicity factors for DDT, dieldrin, and mirex. Chronicity factors have been determined for so few compounds that their interpretation remains somewhat in doubt. However, some guidance can be obtained from the fact that the chronicity factors for caffeine, table salt, and the few organic phosphorus compounds studied so far are all between 0.5 and 1.5. The ability of the body to detoxify cyanide is tremendous, and the chronicity factor of potassium cyanide is less than 0.04. On the contrary, the chronicity factor of warfarin is 21, while that of hexamethylphosphoramide is greater than 200. It is difficult to evaluate the significance of values in the order of 5 such as that of DDT. However, chronicity factors greater than 10 indicate considerable accumulation of toxic effect. The chronicity factor of mirex is unusually high and would seem to indicate caution in use of that compound.

Degree of Cumulation of Storage

The relation of cumulative toxic effects to cumulation of storage can be conveyed most clearly after mentioning the concentration index. This is true even though this index has not been widely used for any class of chemicals and, without additional information, can only be estimated for the compounds under study.

All compounds reach higher tissue levels when administered repeatedly at a sufficiently short interval than when administered

only once at the same rate. However, the degree of increase varies greatly for different compounds. The *concentration index* (R_c) has been proposed by Wagner (1967) as a quantitative measure of the degree of build-up. The index, which is expressed as a quotient, is defined as a ratio between the average blood concentration at equilibrium and the average blood concentration after a single dose. Since the concentration index is a ratio, it may be used to compare the tendency of different compounds to accumulate in the tissues regardless of the different ranges of dosage at which they are tolerated.

A particular tissue level of each toxicant has its characteristic effect no matter what schedule of dosage produced the concentration. Of course, the toxic effect persists insofar as the compound or its toxic metabolites accumulate. However, some compounds with low concentration indices have highly cumulative effects as measured by their chronicity factors. Persistence of effect is found in connection with sufficient absorption of compounds that are stored effectively, but it is not limited to such compounds. Thus, the two phenomena represented by the chronicity factor and the concentration index must not be confused. Measurement of both of them for a wide range of compounds would certainly help to clarify our understanding of toxicology.

Pathology

Even repeated doses of chlorinated hydrocarbon insecticides do not produce pathology sufficient to account for death or form the basis for diagnosis. It is true that certain changes in the livers of rats have been spoken of as "characteristic." However, very similar changes are produced in the liver by chemically unrelated compounds, yet none of the compounds produce the lesions in non-rodents. The changes under discussion involve enlargement of liver cells accompanied by migration of the coarse basophilic cytoplasmic granules to the periphery of the cell, an increase in fat, and the presence of cytoplasmic inclusion bodies of complex structure sometimes called lipospheres. It has been shown that these inclusion bodies consist of fatty droplets surrounded by walls composed of smooth endoplasmic reticulum arranged in a laminar way. There is considerable reason to believe that these inclusion bodies are a visible evidence of a defense mechanism. They occur in animals that thrive in spite of continued heavy dosage and not in animals that succumb to the same or even higher dosage levels. The

question of whether liver changes represent adaptation or injury is best decided in terms of microsomal enzyme function rather than in terms of these reversible morphological changes (Hutterer *et al.*, 1968).

TOXICITY TO MAN

Experimental

Apparently only three of the chlorinated hydrocarbon insecticides, DDT, dieldrin, and methoxychlor, have been studied experimentally in man. None of the dosages given have produced any clinical effect. The highest ones administered are shown in Table 3, which also shows the duration of treatment and the highest average storage observed in body fat.

TABLE 3 – Maximal dosages of chlorinated hydrocarbon insecticides that have been given to volunteers (all without clinical effect) and the maximal average storage of these compounds in fat resulting from these exposures.

Compound	Dosage (mg/kg/day)	Duration (days)	Storage (ppm)	Reference
DDT (Tech)	0.5	654	281	Hayes, unpublished
p,p′-DDT	0.5	654	325	Hayes, unpublished
Dieldrin	0.03	749	3.62	Hunter *et al.*, 1969
Methoxychlor	2.0	56	–	Stein *et al.*, 1965.

Use Experience

Workers heavily exposed to DDT for as much as 19 years absorbed about half as much DDT each day as volunteers dosed at the rate of 0.5 mg/kg/day. Careful study of these workers has failed

to reveal any illness attributable to their exposure (Laws *et al.*, 1967).

Workers showing fat levels of dieldrin as high as 32 ppm and blood levels as high as 0.137 ppm have remained well (Hayes and Curley, 1968). However, workers with greater exposure who developed somewhat higher storage levels have shown convulsions and other signs typical of poisoning. Dieldrin is apparently the only chlorinated hydrocarbon insecticide that has led to poisoning in workers except under very exceptional conditions. As mentioned earlier, some episodes of occupational poisoning by benzene hexachloride have not recurred in other situations in which the exposure was presumably as great, thus suggesting involvement of some unusual factor.

Accidental Poisoning

Most poisoning caused by chlorinated hydrocarbon insecticides has been associated with ingestion, sometimes resulting from gross

TABLE 4 – Summary of epidemics of poisoning by pesticides. Partly from Weeks (1967).

Kind of Accident	Pesticide Involved	Material Contaminated	Number of Cases	Number of Deaths
Spillage During Transport or Storage	Endrin	Flour	159	0
	Endrin	Flour	3	0
	Endrin	Flour	874	26
	Dieldrin	Food	21	0
	Parathion	Wheat	360	102
	Parathion	Barley	38	9
	Methyl para.	Sugar	300	16
	Parathion	Sheets	3	0
	Mavinphos	Pants	6	0
Eating Formulation	Hexachloro-benzene	Seed-grain	>3,000	3-11%
	Organic mercury	Seed-grain	34	4
			321	35
	Warfarin	Salt	14	2
Improper Application	Toxaphene	Collards	7	0
	Nicotine	Mustard	11	0
Miscellaneous	Parathion	Crops	354	0

contamination of food (Weeks, 1967), sometimes resulting from the availability of poisons to children (See Table 4). Some of the most dramatic outbreaks were associated with endrin, the most acutely toxic of the chlorinated hydrocarbon insecticides in common use. It is interesting, however, that endrin is excreted more rapidly than many of the other compounds so that it disappears rapidly from the blood and tissues of persons who survive poisoning by it. This rapid excretion apparently accounts for the fact that occupational poisoning by endrin is unusual. The compound could not be detected in the blood and fat of men working in a factory where it is manufactured and formulated (Hayes and Curley, 1968).

FIGURE 3 – Relation of dietary intake of DDT and dieldrin and their storage in human fat. Modified from Durham *et al*. (1965).

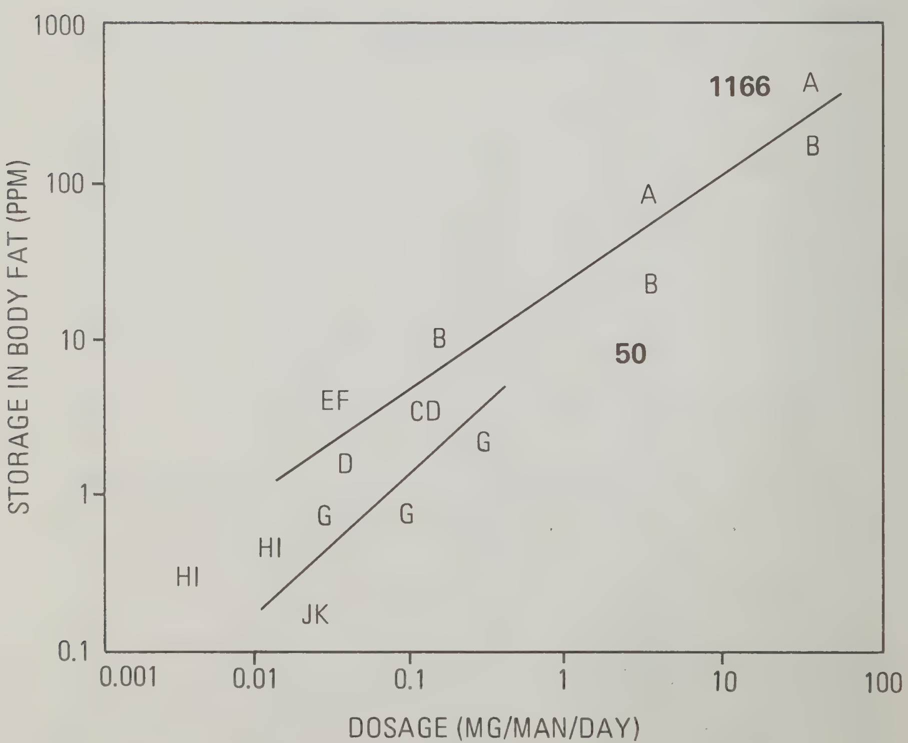


TABLE 5 – Concentration (ppm) of DDT and DDE in fat of people. Modified from Hayes (1966).

CONCENTRATION (PPM) OF DDT AND DDE IN FAT OF PEOPLE

EXPOSURE GROUP	DDT	DDE
1. DIED BEFORE DDT	0	0
2. ABSTAIN FROM MEAT	2.3	3.2
3. GENERAL POPULATION	4.9	6.1
4. ENVIRONMENTAL EXPOSURE	6.0	8.6
5. AGRICULTURAL APPLICATORS	17.1	22.3
6. FORMULATORS	162	91
7. VOLUNTEERS (35 mg/day)	281	36
8. ESKIMOS	0.8	2.0

TABLE 6 – Mean storage of insecticides (ppm in fat). Modified from Hayes (1966).

	U.S.A.	ENGLAND & WALES	INDIA (Delhi)
DDT	1.79	1.1	14.29
DDT + DDE	10.32	3.3	30.20
DDD	0.45		2.70
BHC	0.60	0.42	1.70
HEPTACHLOR EPOXIDE	0.24	$\lesssim$ 0.10	< 0.03
DIELDRIN	0.29	0.26	0.03

Laboratory Findings

The storage of chlorinated hydrocarbon insecticides is dose-related whether exposure is oral (Figure 3) or occupational (Table 5).

This storage has been reported from at least 13 countries and presumably occurs in all countries (See Table 6).

Some persons employed in insecticide factories as clerks or secretaries are not exposed to the compound any more than are people in the general population. However, in spite of some overlapping of the values, one can generally distinguish storage levels in persons under the following four classifications: (a) persons without special exposure, (b) healthy formulators and applicators with heavy exposure, (c) persons who are poisoned but survive, and (d) persons killed by poisoning. Known values are shown in Table 7. It is important to remember that various drugs and especially arsenic, mercury, and lead are found commonly in human tissue. It is not the mere presence of foreign chemicals but their concentration that determines their effect or lack of it.

In the United States the concentration of DDT stored by the general population did not increase after it was first measured in 1950, and it may have decreased somewhat in recent years (see Table 8).

CRITIQUE OF SAFETY

The only assurance we have for the safety of pesticides, drugs, or foods is the dosage-response relationship. Even materials of low toxicity such as aspirin or materials that are necessary to life such as salt or water can produce severe intoxication or death if misused.

Under present law, pesticides can be applied to crops or to animals that produce our food only after extensive studies in animals have given assurance that the amount of the compounds that we may eventually ingest will be safe (Anderson, 1966; Kirk, 1966). However, because species frequently differ in their susceptibility to chemicals, it is wise to monitor their effects in man, particularly when they are first introduced. In fact, as summarized in Table 9, the factor of species may introduce greater variation in the toxicity of a compound than any other factor except dosage (Hayes, 1967a).

TABLE 7 – Storage of chlorinated hydrocarbon pesticides in the general population of the United States, in exposed workers, and in patients who survived poisoning or failed to do so.

Sample and Compound	General Pop. (ppm)	Workers (ppm)	Patients (ppm)	Dead (ppm)
Body fat except as noted for fatal cases				
p,p′-DDT	0.90 - 4.97	18.12 -283.83		19-36 H, K, & L
p,p′-DDE	0.48 -21.17			
r -BHC	N.D.<0.30			343; 89L
dieldrin	0.02 - 1.15	0.60 - 31.96	48	
aldrin	N.D.<0.01	N.D.<0.01	45 as dieldrin	
endrin	N.D.<0.03	N.D.<0.03		0.6-0.7 K&B;7.2L 0.11-0.69 K&L
heptachlor e.	0.03 - 1.45			78.0L,
toxaphene	N.D.<0.03			6.75-14.03 K,L&B
Blood				
p,p′-DDT	0.0008-0.0490	0.0271-0.9955		
p,p′-DDE	0.0024-0.0416	0.0315-0.8546		
r -BHC	<0.0003-0.0132			
dieldrin	<0.0001-0.0129	<0.0007-0.1370	0.28	0.65
aldrin	N.D.<0.0001	<0.0001-0.0039	0.036 as aldrin 0.28 as dieldrin	
endrin	N.D.<0.0001	N.D.<0.004	<0.004-0.053	
heptachlor e.	<0.0002-0.0051			
toxaphene			0.156	10
Urine				
p,p′-DDT	0.0018-0.0138	0.0023-0.0238		
p,p′-DDE	0.0114-0.0285			
r -BHC	<0.0001-0.0004			
dieldrin	0.0005-0.0019	0.0013-0.0660	0.081	
aldrin	N.D.<0.0001	N.D.<0.0002		
endrin	N.D.<0.0001	N.D.<0.004	<0.004-0.039	
heptachlor e.	<0.0001-0.0009			
toxaphene	N.D.<0.0001			

H = heart; K = kidney; L = liver; B = brain; N.D. = searched for but not found.

TABLE 8

Concentration of DDT-Derived Material in Body Fat of the General Population of the United States.
After Hayes (1966).

Year	Location	No. of Samples	Analysis Method	DDT (ppm)	DDE as DDT (ppm)	Total as DDT (ppm)	DDE as DDT (% of total)
<1942	Louisville, Ky.	10	Colorimetric	[a]	[a]	[a]	–
1950	Washington, D.C.	75	Colorimetric	5.3	–	5.3	–
1955	Tallahassee, Fla.	49	Colorimetric	7.4	12.5	19.9	63
1954-1956	Savannah, Ga. and Wenatchee, Wash.	61	Colorimetric	4.9	6.8	11.7	58
1956	Atlanta, Ga.	36	Colorimetric	5.5	10.1	15.6	65
1961-1962	Atlanta, Ga., Louisville, Ky., Phoenix, Ariz., and Wenatchee, Wash.	130	Colorimetric	4.0	8.7	12.7	69
1961-1962	Wenatchee, Wash.	28 [b]	GLC [c]	2.4	4.3	6.7	64
1962-1963	Chicago, Ill.	282	GLC [c]	2.9	8.2	11.1	74
1964	Northeast, Midwest, Deep South, and Far West	64	GLC [c]	2.5	5.1	7.6	67
1964	New Orleans, La.	25	GLC [c]	2.3	8.0	10.3	77

a Not detected.

b These 28 samples were also tested for DDT and DDE content by a colorimetric method. These results are included in the 130 samples listed above.

c Gas-liquid chromatography.

TABLE 9 – Summary of information on the importance of different factors (other than dosage) influencing toxicity. After Hayes (1967a).

Factor	Total No. of Compounds	Ratio of Difference: Range	Ratio of Difference: Mean	Increasing Ratio Indicates
Route	67	0.2 - 21	4.2	oral >[a] dermal
Species	20	0.2 - 11.8	1[b]	other sp. >rat
	1[c]	>230		other sp. >rat
Individual[d]				
oral route	69	1.20 - 7.14	2.42	$LD_{50} > LD_1$
dermal route	42	1.37 - 14.93	3.00	$LD_{50} > LD_1$
Sex				
oral route	65	0.21 - 4.62	0.94	male > female
dermal route	37	0.11 - 2.93	0.81	male > female
Age	18	0.6 - 10.0	2.9	newborn >adult
	16	0.7 - 6.2	––	newborn >adult
	15	0.2 - 4.1	1.8	newborn >adult
Duration	22	0.5 - 20.0	––	2-year >90-day

a. " > " indicates greater toxicity of chemical or greater susceptibility of animal; b. Approximate value; c. Norbormide; d. Same sex.

Studies in man may be made in volunteers or in men with intensive and prolonged occupational exposure. Each kind of study has its own special advantages, but both contribute information on the possible effects of relatively high and sustained rates of absorption (Hayes, 1968).

Studies of volunteers (See Table 3) and of workers (see section on use experience) demonstrate the safety of (a) methoxychlor, (b) DDT except in connection with ingestion, and (c) dieldrin except in connection with ingestion and some but not all occupational situations. Volunteers have been safely fed DDT at a dietary rate over 900 times greater than that of the general population (Hayes *et al.*, 1956; Durham *et al.*, 1965). Dieldrin has been safely fed at a

dietary rate about 27 times greater than that of the general population.

In considering the future safety of workers who continue to be employed in the formulation of DDT, one must depend largely on animal experiments. Rats withstand a daily dosage at least 10 times that of heavily exposed workers without any detectable clinical effect (Fitzhugh and Nelson, 1947; Ortega *et al.*, 1956), although minimal reversible tissue change may be present (Ortega *et al.*, 1956). Dogs (Lehman, 1952) and monkeys (Durham *et al.*, 1963) also withstand a daily dosage 10 times greater than that of the workers, but they do not show the tissue changes, which seem to be peculiar to rats. Although one can not predict the future safety of workers from their present tolerance for DDT, one can use the experience already gained to predict the future safety of the general population in relation to DDT. It has been shown for at least two animal species that toxicity resulting from a lifetime of exposure is seldom very different from toxicity resulting from 90 days of exposure at the same dosage rate (Weil and McCollister, 1963). The largest factor of difference observed was 20. Ninety days constitute about one-eighth of the life-span of a rat. Many workers have now been exposed to DDT for much more than one-eighth of their life-span. Since they have not suffered detectable harm, it seems most unlikely that the general population will be harmed by dosages more than 900 times smaller than those to which the workers are exposed.

In general, this same argument holds for dieldrin. Workers tolerate this compound at an average of about 1.5 times greater than that fed to volunteers and about 40 times greater than that of the general population (Hayes and Curley, 1968). However, the occurrence of occupational poisoning in other situations suggests that intake of dieldrin at a rate 150 times greater than that of the general population is dangerous.

The chlorinated hydrocarbon insecticides, especially DDT, have a good safety record although they are tenaciously stored. As they are replaced by compounds that are less stored, there may be an increased hazard to workers. There has already been an increase in deaths caused by the more toxic organic phosphorus insecticides. As really new compounds or other means of pest control are introduced, it must be remembered that mere lack of persistence coupled with low acute toxicity can not always be equated with safety.

CONCLUSIONS

The chlorinated hydrocarbon insecticides differ greatly in their toxicity and in their persistence in the mammalian body once they have been absorbed. All of their effects are dose-related. However, species differ in their susceptibility and also in their access to the compounds under any particular pattern of use.

The chlorinated hydrocarbon insecticides also differ greatly in the contribution they have made and continue to make to the control of human disease and the solution of other specific problems. It would be a grave mistake to treat these compounds as identical merely because they share the same group name and certain broad chemical features. It would also be a mistake to suppose that other compounds are necessarily safe because they are not persistent. After all, if a perfect substitute for the chlorinated hydrocarbon insecticides were available, it would be adopted without the necessity of a conference.

REFERENCES

Anderson, R. J.: Official Registration of Pesticides in *Scientific Aspects of Pest Control*, National Academy of Sciences-National Research Council Publication 1402, pp. 385-391, 1966.

Brown, R. R., Miller, J. A., and Miller, E. C.: The Metabolism of Methylated Aminoazo Dyes. IV. Dietary Factors Enhancing Demethylation *in vitro*. *J. Biol. Chem. 209:*211-222, 1954.

Burlington, H., and Lindeman, V. F.: Effect of DDT on Testes and Secondary Sex Characters of White Leghorn Cockerels. *Proc. Soc. Exp. Biol.*, New York, *74*(1):48-51, 1950.

Conney, A. H.: Pharmacological Implications of Microsomal Enzyme Induction. *Pharmacol. Rev. 19:*317-366, 1967.

Dale, W. E., Gaines, T. B., and Hayes, W. J., Jr.: Storage and Excretion of DDT in Starved Rats. *Toxicol. Appl. Pharmacol. 4:*89-106, 1962.

Dale, W. E., Gaines, T. B., Hayes, W. J., Jr., and Pearce, G. W.: Poisoning by DDT: Relation Between Clinical Signs and Concentration in Rat Brain. *Science, 142:*1474-1476, 1963.

Durham, W. F.: The Interaction of Pesticides with Other Factors. *Residue Rev. 18:*21-103, 1967.

Durham, W. F., Ortega, P., and Hayes, W. J., Jr.: The Effect of Various Dietary Levels of DDT on Liver Function, Cell Morphology, and DDT Storage in the *Rhesus* monkey. *Arch. Intern. Pharmacodyn. 141*:111-129, 1963.

Durham, S. F., Armstrong, J. F., and Guinby, G. E.: DDT and DDE Content of Complete Prepared Meals. *Arch. Environ. Health., 11:*641-647, 1965.

Ferguson, J.: The Use of Chemical Potentials as Indices of Toxicity. *Proc. Roy. Soc. B., 127:*387, 1939.

Fitzhugh, O. G., and Nelson, A. A.: The Chronic Oral Toxicity of DDT (2, 2-Bix-p-chlorophenyl-1, 1, 1-trichloroethane). *J. Pharmacol. 89:*18-30, 1947.

Gaines, T. B.: Toxicology of Pesticides. *Toxicol. Appl. Pharmacol.* (In Press).

Gaines, T. B., and Kimbrough, R. D.: The Oral Toxicity of Mirex in Adult and Suckling Rats. (In Press).

Garrettson, L. K., and Curley, A.: Dieldrin: Studies in a Poisoned Child. (In Press).

Hayes, W. J., Jr.: Review of the Metabolism of Chlorinated Hydrocarbon Insecticides Especially in Mammals. *Ann. Rev. Pharmacol. 5:*27-52, 1965.

Hayes, W. J., Jr.: Monitoring Food and People for Pesticide Content. Scientific Aspects of Pest Control. NAS-NRC Publ. No. 1402, pp. 314-342, 1966.

Hayes, W. J., Jr.: Toxicity of Pesticides to Man: Risks from Present Levels. *Proc. Royal Soc. B., 167:*101-127, 1967a.

Hayes, W. J., Jr.: The 90-Dose LD_{50} and a Chronicity Factor as Measures of Toxicity. *Toxicol. Appl. Pharmacol. 11:*327-335, 1967b.

Hayes, W. J., Jr.: Tests in Man. Chap. 10 in *Modern Trends in Toxicology.* E. Boyland and R. Goulding (Ed.), Butterworths, London, pp. 198-230, 1968.

Hayes, W. J., Jr., and Curley, A.: Storage and Excretion of Dieldrin and Related Compounds. *Arch. Environ. Health, 16:*155-162, 1968.

Hayes, W. J., Jr., and Dale, W. E.: Concentration of DDT in Brain and Other Tissues in Relation to Symptomatology. *Toxicol. Appl. Pharmacol. 6:*349, 1964.

Hayes, W. J., Jr., Durham, S. F., and Cueto, C., Jr.: The Effect of Known Repeated Oral Doses of Chlorophenothane (DDT) in Man. *J.A.M.A. 162*(9):890-897, 1956.

Heath, D. F., and Vandekar, M.: Toxicity and Metabolism of Dieldrin in Rats. *Brit. J. Ind. Med. 21:*269-279, 1964.

Hunter, C. G., Robinson, J., and Roberts, M.: Pharmacodynamics of Dieldrin (HEOD). *Arch. Environ. Health, 18*(1):12-21, 1969.

Hutterer, F., Schaffner, F., Klion, F. M., and Popper, H.: Hypertrophic, Hypoactive Smooth and Endoplasmic Reticulum: A Sensitive Indicator of Hepatotoxicity Exemplified by Dieldrin. *Science, 161:*1017-1019, 1968.

Kirk, J. K.: The Role of the Food and Drug Administration in Regulation of Pesticides. Scientific Aspects of Pest Control, NAS-NRC Publ. 1402, pp. 392-398, 1966.

Kupfer, D.: Effects of some Pesticides and Related Compounds on Steriod Function and Metabolism. *Residue Rev. 19:*11-30, 1967.

Laws, E. F., Jr., Curley, A., and Biros, F. J.: Men with Intensive Occupational Exposure to DDT. A Clinical and Chemical Study. *Arch. Environ. Health, 15:*766-775, 1967.

Lehman, A. J.: Chemicals in Foods; A Report to the Association of Food and Drug Officials on Current Developments. Part II. Pesticides. *Quart. Bull. Assoc. Food and Drug Officials of U.S. 15:*122-133, 1951.

Lehman, A. J.: Chemicals in Foods: A Report to the Association of Food and Drug Officials on Current Developments. Part II. Pesticides. Section II. Dermal Toxicity; Section III. Subacute and Chronic Toxicity; Section IV. Pathology. *Assoc. Food and Drug Officials U.S. Quart. Bull.* 16, 3, 47, 126, 1952.

Ortega, P., Hayes, W. J., Jr., Durham, W. F., and Mattson, A.: DDT in the Diet of the Rat. *Public Health Monograph No. 43,* PHS Publ. No. 484, p. 27, 1956.

Remmer, H.: Die Beschleunigung des Evipanabbaues unter der Wirkung von Barbituraten. *Naturwissenschaften, 45:*189-190, 1958a.

Remmer, H.: Die Wirkung der Nebennierenrinde auf den Abbau von Pharmaka in der Lebermikrosomen. *Naturwissenschaften, 45:*522-523, 1958b.

Robinson, J. and Hunter, C. G.: Organochlorine Insecticides: Concentrations in Human Blood and Adipose Tissue. *Arch. Environ. Health, 13:*558-563, 1966.

Stein, A. A., Serrone, D. M., and Coulston, F.: Safety Evaluation of Methoxychlor in Human Volunteers. *Toxicol. Appl. Pharmacol. 7:*499, 1965.

Wagner, J. G.: Drug Accumulation. *J. Clin. Pharmacol.* March-April, 84-88, 1967.

Weeks, D. E.: Endrin Food-Poisoning. *Bull. Wld. Hlth Org. 37:*499-512, 1967.

Weil, C. S., and McCollister, D. D.: Relationship Between Short- and Long-term Feeding Studies in Designing an Effective Toxicity Test. *J. Agr. Food Chem. 11:*486-491, 1963.

APPENDIX III

Health Effects from DDT Norfolk County

R. B. Sutherland, M.D., C.M., D.P.H., D.I.H.
Chief, Health Studies Service
Environmental Health Services Branch
Ontario Department of Health

During 1968, approximately 40 per cent of all DDT sold in Ontario in quantities of 160 fluid ounces or 4 pounds or more was for application in Norfolk County. Comparative data for earlier years are not available, but it can be assumed that for some years this County has probably received relatively large proportions of the DDT sold.

No human cases of disease of any kind have been reported in the literature from chronic ingestion of DDT. Based upon animal experimentation, it has been suggested that injury to the liver and kidneys might be looked for in humans. In addition, one might consider the possibility of central nervous system effects.

No sources of morbidity information relating to Norfolk County are immediately available, and arrangements for this kind of data would have to be set up as a special study. However, certain mortality data for this County are available in the annual reports on Vital Statistics from the Department of the Registrar General. These data are classified by sex (though not by age group) in rather broad diagnostic groups, known as the "A" list of the International Statistical Classification of Diseases, Injuries and Causes of Death. The following tables present the numbers of deaths, for men and women separately, that occurred during the two 3-year periods 1960-1962 and 1965-1967 from certain "A" list diagnostic groupings that might possibly be expected to show an increase in incidence if the intake of DDT during the past decade were in fact contributing to mortality in Norfolk County.

For four of the diagnostic groupings, the numbers of recorded deaths were sufficiently large that it was considered worth while to

DEATHS FROM SELECTED CAUSES
NORFOLK COUNTY

Cause of Death		Male		Female	
		1960-62	1965-67	1960-62	1965-67
A101	Gastritis & Duodenitis	0	0	2	0
A102	Appendicitis	1	0	0	1
A104	Gastroenteritis & Colitis	3	2	4	3
A105	Cirrhosis of Liver	5	7	2	6
A108	Acute Nephritis	1	0	0	0
A109	Chronic & Other Nephritis	4	5	5	4
A110	Infections of Kidney	3	8	2	1
A 71	Non Meningococcal Meningitis	0	1	2	2
A 72	Multiple Sclerosis	2	0	0	1
A 73	Epilepsy	2	1	0	0

compute the numbers of deaths from these causes that would "normally" have been expected in each sex in Norfolk County during the two 3-year periods. Average annual age-, sex- and cause-specific provincial death rates for each of the 3-year periods were derived by taking the average number of deaths in the province in each age and sex group during each 3-year period, dividing these averages by the respective numbers of males and females in each age group in the Ontario population, and multiplying each quotient by 100,000. To estimate the County population during each 3-year period, the numbers of males and females in each age group at the time of the 1961 and 1966 census were multiplied by three. The numbers of expected deaths from each cause were then calculated by multiplying the numbers of males and females in each age and sex group of the County population during each 3-year period by corresponding age- and sex-specific death rates for the province, and summing the products for each sex in each 3-year period. These sums represent the numbers of deaths from each cause that would have

occurred in the County males and females over each 3-year period had the Ontario age- and sex-specific mortality rates applied, and may be compared directly with the number of deaths from these causes that were actually recorded.

As a further check, a similar comparison was made of recorded and expected mortality during the same 3-year periods for males and females in the City of London, a community where we may assume that very little DDT has been applied. The comparisons of recorded and expected mortality for Norfolk County and the City of London are set out in the following table.

The statistical significance of differences between recorded and expected mortality was assessed by calculating the 95 per cent confidence limits for the various numbers of expected deaths, from expansion of the Poisson distribution. In no instance was the number of recorded deaths significantly greater or less than the number expected. That is, there is no evidence of the occurrence of any differences larger than might reasonably be attributed to chance variation in mortality.

COMPARISON OF RECORDED AND EXPECTED MORTALITY FROM SELECTED CAUSES

NORFOLK COUNTY

		Male		Female	
		1961	1966	1961	1966
Population (All Ages)		25,675	25,597	24,800	24,981
Cause of Death		**1960-62**	**1965-67**	**1960-62**	**1965-67**
Gastro-enteritis	Rec.	3	2	4	3
& Colitis	Exp.	3.19	2.55	2.86	2.68
Cirrhosis of	Rec.	5	7	2	6
Liver	Exp.	7.49	8.49	4.02	4.63
Chronic	Rec.	4	5	5	4
Nephritis	Exp.	5.07	4.21	3.58	2.99
Infections	Rec.	3	8	2	1
of Kidney	Exp.	3.25	2.86	2.48	2.73

CITY OF LONDON

		Male		Female	
		1961	1966	1961	1966
Population (All Ages)		82,618	94,294	86,951	100,122
Cause of Death		**1960-62**	**1965-67**	**1960-62**	**1965-67**
Gastro-enteritis	Rec.	9	9	13	12
& Colitis	Exp.	9.7	8.23	10.61	10.15
Cirrhosis of	Rec.	16	35	15	16
Liver	Exp.	21.72	26.22	14.28	17.17
Chronic	Rec.	11	7	8	11
Nephritis	Exp.	15.65	13.58	13.52	11.48
Infections	Rec.	13	9	10	15
of Kidney	Exp.	9.60	8.94	9.34	10.44

APPENDIX IV

Report on Background Data on Use of DDT and its Adverse Effect on Fish and Wildlife

A. E. Armstrong
Fish and Wildlife Branch
Ontario Department of Lands and Forests

Since its development during World War II, DDT has been used extensively for pest control purposes. Its widespread use has provided many opportunities for comprehensive studies of its persistence and its effects, both lethal and sub-lethal.

In Canada and the U.S.A., DDT has been responsible for extensive fish kills, causing great damage to fisheries. This compound and its breakdown products have accumulated in the aquatic environment, producing eventual death of fish and aquatic insects or causing sub-lethal effects such as impairment of fish reproduction. DDT has been detected at great distances from its point of application. It has been found in fish that live far out at sea, in ground waters, fresh-water fish, migratory birds, wild mammals, humans, and in processed dairy food products in many parts of the world.

THE CHLORINATED HYDROCARBONS: DDT, ITS PERSISTENCE AND USE IN THE AQUATIC ENVIRONMENT

The chlorinated hydrocarbons include a large number of chemicals with high insecticidal activity. They are especially resistant to degradation to non-toxic end products and may persist for months or years following application. Chlorinated organic insecticides can contaminate surface waters by run off from treated crop lands or forest areas, by the direct application to bodies of water as in mosquito control, and by the coincidental covering of surface waters during airplane spraying of adjacent land areas.

DDT is practically insoluble in water, dilute acid and alkalies. It is readily soluble in many organic solvents. Since it has a low volatility and is not normally decomposed by sunlight, it possesses a

high residual capacity. In the soil, DDT is very stable and decomposes at a rate of about five per cent per year depending upon soil type. Although relatively insoluble in water, DDT may be carried by solids for long distances in streams and lakes. It is absorbed rapidly from the water by fish, and it has been shown that fish can accumulate hundreds of parts per million, particularly in the fat. In Canada, a residue tolerance limit of 7 ppm DDT has been established by the National Health and Welfare Department for fat of cattle, hogs and sheep[5].

EFFECTS OF DDT ON AQUATIC FAUNA

A considerable amount of literature has been published on the effects of DDT on aquatic life. Many of the studies have been conducted in areas where the application of DDT resulted in direct mortality to fish or wildlife.

Perhaps the most familiar investigation to fisheries personnel in Ontario is that conducted at Lake George, New York. In 1955, the fish hatchery at Lake George, New York, experienced a complete loss of fry from more than 347,000 eggs taken in 1955 from lake trout in Lake George. The loss was characterized by a distended air bladder and air in the intestinal tract, with these symptoms appearing after absorption of the yolk sac when the fry were about ready to feed. Pathological examination failed to show the presence of any disease.

The same situation occurred with eggs collected in 1956. In 1957 and 1958, the eggs collected from Lake George were distributed to three different hatcheries. No survival occurred in 1957, and a negligible survival in 1958. Eggs obtained from Lake George females crossed with males from other lakes failed to survive. Normal survival occurred when females from other lakes were crossed with Lake George males.

Since 1951, extensive quantities of DDT had been used in the Lake George watershed for gypsy moth and black fly control. In 1959, a preliminary study showed that fry from Lake George and other waters where fry were similarly affected contained measurable quantities of DDT and its metabolites. Based on the relationship of the concentration of DDT to the development of the syndrome, it is concluded that the fry mortality is induced by DDT. It occurs when the Schechter-Heller procedure indicates a concentration in the ether extract of the egg equivalent to about 2.9 ppm or above in the weight of the fry[3].

In New Brunswick, large forest areas have been sprayed with DDT at a rate of 0.5 lbs/acre in efforts to control spruce budworm. Several streams in the area were subsequently studied. In 1954, all stations sampled on the Northwest Miramichi River were within the area sprayed with DDT for the first time. The drastic effects of the spray were clearly indicated when not one fry was found that year. Small and large parr were also reduced, but to a lesser extent than the fry.

Direct evidence of the harmful effects of the DDT was obtained in 1954 by holding salmon parr in cages in several parts of the Northwest Miramichi. From 63 per cent to 91 per cent of those held within the spray area were dead in three weeks, while only two per cent died in an unsprayed control stream during the same period. Laboratory bioassays conducted in 1957 showed that the median tolerance limit (concentration killing one half a sample of fish) for small parr in DDT was 0.049 ppm for a 24-hour period, and 0.047 ppm for 48 hours[10].

A study of the effect of DDT spraying on aquatic insects was also conducted on the Miramichi River in New Brunswick. The extreme reduction in the aquatic stages of larger insects such as mayflies, stoneflies and caddisflies was apparent, and from the standpoint of the fish created a serious decline in the bulk of fish food. Another and perhaps the most serious consequence of the spraying is that many of the insects which were eliminated have not become re-established even in the second year after spraying[8].

In 1957, an aerial spraying program was conducted on 155,000 acres of timberland on northern Vancouver Island, in an attempt to control an outbreak of black-headed budworm. A rate of 1 lb of DDT/acre was used. The damage to the fish and fish-food populations was assessed on the major streams and, on four of these, was found to be severe. The fish mortality was confined generally to cohoe fry, trout, steelhead yearlings and possibly alevins of both trout and steelhead. In the four major streams affected by spraying, the progeny of an estimated 1956 escapement of 43,000 cohoe adults and the juvenile stages of several thousand steelhead and trout was almost eliminated[7].

In the summer of 1955, a considerable area of forest along the Yellowstone River in Montana was sprayed with DDT in oil at the rate of 1 lb/acre to control spruce budworm. An estimated 99 per

cent of the aquatic insects along a 100-mile section of the stream were killed. Losses of trout, whitefish and suckers appeared in early October, three months after the spraying. It is believed that many fish died of starvation because of the high mortality of aquatic insects caused by the spraying[6].

It is the view of many people that the long-term effects, the sub-lethal effects, on non-target species are of far greater significance than direct mortalities. Studies on the accumulation of DDT in fish, and its sub-lethal effect, have been conducted recently in the State of New York. The DDT content of 16 species of fish taken from New York waters was found to range from 0.2 to 7.0 ppm DDT on a fresh weight basis. Certain tissues, such as visceral fat, gills, eggs and the reproductive organs, contained up to 40 ppm. The surrounding water and sedimentary mud contained much smaller amounts, indicating that the fish had concentrated considerable amounts of DDT[11].

In spite of conflicting evidence on the direct effects of DDT applications on aquatic life, the fact remains that DDT persists and accumulates in the environment. As West[17] stated: "The effects of pesticides can be both direct and indirect. The indirect effects are the most disturbing. The most widely used insecticides, the chlorinated hydrocarbons, are concentrated by living matter in the aquatic environment and each link in the food chain presents an increasing build-up of the pesticide. These effects are insidious and are usually not discernible for a long period after initial contact with the pesticide."

The water of a farm pond treated with .02 ppm DDT did not yield a detectable residue three weeks later. Within four weeks, however, fish in the pond had accumulated 3 to 4 ppm DDT and its metabolites DDD and DDE. From 2 to 3 ppm of these metabolites were still present in the fish 17 months after the application[1].

Buckley[2], in New York State, has shown that a diet of ground fish containing 10 ppm of DDT gave rise to a level of 135 ppm in captive bald eagles. The bald eagle population has declined seriously in recent years. It is not possible to prove that this is the direct result of the ingestion of insecticides, but there is no evidence to the contrary.

The spraying for Dutch elm disease at Michigan State University began in 1954. The following spring, dead and dying robins began to

appear on campus. The pattern was repeated with monotonous regularity in succeeding springs. A key piece in the jig saw puzzle of the doomed robins was supplied by Dr. Roy Barker who traced the intricate cycle of events by which the robin's fate is linked to the elm trees by way of the earthworms [12].

Another characteristic is the synergistic effect of some of the chemicals when they occur together in an animal. Research at the Patuxent Wildlife Research Center has disclosed that DDT and 2,4-D fed together had several times greater toxicity to mallard ducks than either fed separately.

As a result of the accumulations of DDT on Lake Simcoe and the Muskoka Lakes system, indications are clear that we should press for the elimination of the use of DDT in these areas as quickly as possible.

PROHIBITION OF DDT AND ALTERNATIVE PESTICIDES

In Michigan, the use of DDT has been discontinued in State Parks. Its use is also avoided in controlling forest pests in State forests. Furthermore, DDT has been virtually eliminated from the recommendations of Michigan State University for control of mosquito larvae. Also, where the University previously recommended DDT as the spray control material for Dutch elm disease, it now recommends methoxychlor.

Aiding greatly in the fight against pesticides in Michigan is Dr. Ralph A. MacMullan, Director of the Michigan Department of Conservation, who wrote in a recent article in the Conservation Agency's bi-monthly magazine: "Michigan has come to a point in its history when it must completely outlaw the use of certain highly destructive pesticides such as DDT, dieldrin, aldrin, heptachlor, endrin, lindane, chlordane, and other 'hard' or persistent chemical compounds used to kill insects."

In New York State, the Conservation Department has a regulation prohibiting the use of DDT on State-owned lands in lake front watersheds. As alternate mosquito larvicides, the State is recommending the use of Malathion – 0.5 lbs/acre, and Methoxychlor – 0.2 lbs/acre.

From information received from the State of Montana, Department of Fish and Game, the use of DDT has been discontinued for

spruce budworm control. Malathion has been recommended as an alternate. The Department of Fish and Game is co-operating on experimental programs aimed at testing new varieties of insecticides for eventual use.

Recently, the Department of Fisheries of Canada has moved into the area of co-operatively assessing the toxicity and comparative hazard presented by chemicals which might be used as substitutes for the highly toxic DDT, which is still favoured as the control agent in large spray programs. Bioassays are conducted concurrently in the field to determine the concentrations which would be acutely toxic to fish and these concentrations are compared with those actually found in the treated area. Dyes are released in the test stream to obtain an indication of the duration of exposure.

Phosphamidon and Dimethoate are relatively new insecticides, and both possess a higher degree of specificity than the DDT which they are intended to replace in British Columbia. Baytex, another relatively new insecticide, was used on quite a large scale with success as a mosquito larvicide in breeding pools adjacent to waters inhabited by fish. These are the insecticides of the future, and are considered better than the broad spectrum insecticides. These new insecticides are costly and their application may take more knowledge. They will likely be accepted and regarded as a step toward the development of highly specific insecticides.

With respect to the use of alternate chemicals, our department has a project for the control of spruce budworm planned for the Port Arthur district, using Phosphamidon and Fenitrothion.

In order to monitor this operation, it is planned that control areas be established as well as areas within the spray zone, and observations will be made before and after spraying so that adequate assessment of the effect on birds, eggs of young, fish, and recovery of aquatic insects, can be studied.

METHODS AND COSTS IN USE OF DDT

The advent of DDT during World War II was the beginning of a drastic change in the character and scope of the problem of pollution by pesticides. New formulations and methods of application were rapidly developed. Dusts, solutions, emulsions, and aerosols were broadcast by ground and air equipment, not only for the control of

insect vestors of disease, but also for the control of forest and agricultural pests.

DDT is applied directly to waters to control mosquito and black fly larvae. For mosquito larvae, DDT is applied as a solution in organic solvents (usually fuel oil) by compressed air sprayers, mist blowers and aircraft. Application rates of 0.25 lbs active ingredient per acre are usually employed. For control of adults, space sprays (0.1 – 0.2 lbs DDT/acre) residual sprays (2 lbs DDT/acre) and aerial sprays (0.25 lbs DDT/acre) are used.

Aerial applications of DDT at 0.25 lbs/acre are also effective for controlling adult black flies. For larviciding, DDT at 0.25 lbs/acre may be used or a solution of DDT in fuel oil can be applied for 15 minutes to a stream at a rate of one part DDT to 10,000,000 parts of water.[4].

It is generally considered that chemical control procedures against mosquitos are most effective and economical when designed to eliminate the larvae. The recommended chemicals are Abate, Fenthion (Baytex), Malathion, Methoxychlor and Pyrethrum.

Adding chemicals to streams to kill the larvae of black flies is the primary control method, and the chemicals considered acceptable for larviciding are Abate and Methoxychlor.

There is no easy method for determining the cost of the use of DDT or alternate chemicals. Most pesticide treatments are generally related to a specific problem, with labour costs often superseding all other costs. In most cases, the cheapest alternate pesticide is still more costly than DDT.

For example, in the use of DDT and an alternate, Methoxychlor, in the control of Dutch elm disease, the costs are \$1.50/gallon and \$4.25/gallon respectively.

DDT has the potential for causing damage; however, for certain control programs it is the only pesticide available. For example, the tobacco grower has no other chemical on which he can rely to control tobacco cutworm.

Following an extensive review of the available literature on the use of DDT, I feel our department has a great responsibility to assure

necessary control of pests without hazard to the environment and its inhabitants, including man.

One way in which we can evaluate the long-term effects of chemicals and their residues in the environment is by monitoring – periodic sampling of the elements of the environment and the animals at fixed locations to determine the load of various chemicals in the environment and animal life. Another necessary activity is "surveillance" – the studies of a specific chemical control operation to learn what happened. Logically, pre- and post-treatment population inventories are usually a necessary part of most surveillance projects.

One means of initiating this monitoring and surveillance would be to have our fish and wildlife biologists routinely check on any extensive insect or weed control projects. In other words, some form of monitoring should be carried on, and steps taken to curtail and eventually eliminate the use of DDT, where it has been determined that it is detrimental to fish and/or wildlife directly or indirectly.

REFERENCES

1. Bridges, W. R., B. J. Kallman, A. K. Andrews. 1963.
   Persistence of DDT and its Metabolites in a farm pond.
   Trans. Amer. Fish. Soc. 92 (4):421-427.

2. Buckley, John L.
   Effects of Pesticides upon Wild Birds and Mammals:
   Pesticides – their use and effect; Proceedings of a Symposium, Albany, New York, Sept. 23, 1963.

3. Burdick, G. E., E. J. Harris, H. J. Dean, T. M. Walker, J. Skea, D. Colby. 1964.
   The accumulation of DDT in lake trout and the effect on reproduction.
   Trans. Amer. Fish. Soc. 93 (2):127-135.

4. Canada Dept. Nat. Defence. 1960.
   Armed Forces manual on pest control. 1960. p. 3-14.

5. Canada Dept. Nat. Health and Welfare. 1965.
   Food and drug directorate. T.I.L. No. 261, p. 14.

6. Cope, Oliver B., and Donald E. Parker. (1956).
   Survey of conditions attending fish mortalities in Yellowstone River following spruce budworm spray project. Report issued by the U.S. Forest Service, File 3, 1956, 9 pp.

REFERENCES *(continued)*

7. Crouter, R. A., E. H. Vernon. 1959.
Effects of black-headed budworm control on salmon and trout in British Columbia.
Can. Fish. Cult. 24: p. 23-40.

8. Ide, P. P. 1960.
Effects of forest spraying with DDT on aquatic insects, food of salmon and trout in New Brunswick.

9. Jackson, K. J.
The Role of the Department of Fisheries of Canada in Dealing with Problems Presented by the Use of Pesticides in British Columbia.
Can. Fish. Cult. 37: p. 45-50.

10. Keenleyside, M. H. A. 1959.
Effect of spruce budworm control on salmon and other fishes in New Brunswick.
Can. Fish. Cult. 24: p. 17-22.

11. Mack, G. L., S. M. Corcoran, S. D. Gibbs, W. H. Gutermann, J. A. Reckhahn, D. J. Lisk. 1964.
The DDT content of some fishes and surface waters of New York State.
*Fish and Game Jour. 11 (2):*p. 147-153.

12. Mehner, John F., and George J. Wallace.
Robin Populations and Insecticides.
Atlantic Naturalist, Vol 14 (1959), No. 1, p. 4-10.

13. Ontario Water Resources Commission.
Unpublished report material and references.

14. Ontario Department of Lands and Forests.
Recommendations For Control Of Mosquitoes And Black Flies In Ontario, 21 pp.

15. Parker, Lansing A.
Fish and Wildlife's Stake in the Pesticide Problem. Given at the 26th Midwest Wildl. Conf., Bloomington, Indiana, Dec. 7, 1964. 15 pp.

16. Tarzwell, C. M. 1959.
Pollutional effects of organic insecticides.
Trans. of 24th N. Amer. Wild life Conf. 1959. p. 132-142.

17. West, I. M. D. 1964.
Pesticides as contaminants.
*Arch. Environ. Health, 9:*p.625-633.

18. Anonymous.
A Rept. of the President's Sci. Advisory Comm., The White House, Washington, D.C., May 15, 1963. 24 pp.

APPENDIX V

Monitoring the Milk Supply in Ontario for Pesticide Residues

R. FRANK and E. H. SMITH

Pesticides are neither a public hazard nor an agricultural problem in Ontario-produced milk. This statement is based on solid scientific findings at the Provincial Pesticide Residue Testing Laboratory at Guelph. One thousand six hundred and fifty-one raw milk samples were tested in a program designed to monitor the milk supply over a 20-month period. Each sample was made up of milk from approximately 16 producers, and included milk from producers of *fluid* and *industrial* milk, which number approximately 18,000 and 7,900, respectively.

Of more than 400 registered pesticide chemicals that are used in agriculture and forestry, less than ten of these can persist in the body tissues of the dairy cow and pass into the milk. Those that do persist are insecticides belonging to a group called chlorinated hydrocarbons, which includes DDT, DDD, aldrin, dieldrin, lindane, heptachlor, and others. DDT, while being stored in the body fat and secreted in the milk fat, is also metabolized to DDD and DDE. The nature of these changes is a reduction in the number of chlorine atoms attached to the molecule. Heptachlor and aldrin are not stored in animal tissues as such, but are metabolized, by the addition of oxygen, to heptachlor epoxide and dieldrin respectively, and these persist in animal fat and appear in the milk. This process is not a detoxification process. The persistence of lindane in body tissues and its subsequent appearance in milk is much shorter termed than the other chlorinated hydrocarbons. Methoxychlor is so rapidly metabolized by the dairy cow that is is rarely found in milk even after spraying the animal.

Since the persistent insecticides are almost entirely confined to the fat portion of all foods, chemical analysis to determine presence or absence of pesticides involves first the separation of the fat from the milk and secondly the extraction and isolation of the pesticides

from the fat with the aid of solvents and absorbants. The individual chlorinated hydrocarbons are separated between gas and liquid phases by gas-liquid-partition chromatography so that each can be identified and the quantity measured down as low as a few parts per billion (ppb). If one part per million (ppm) is one cent in ten thousand dollars, then one part per billion (ppb) is one cent in ten million dollars.

The average individual in India is carrying about 37 ppm of DDT in his tissues with no apparent detriment to his well-being. The average Canadian carries around 10 ppm.

During the sixties, the Governments of several countries have been meeting under the auspices of FAO/WHO to establish practical residue limits for foodstuffs shipped internationally. In milk fat, the 1969 levels of 1.25 ppm total DDT, 0.2 ppm lindane, 0.125 ppm aldrin plus dieldrin and .025 ppm heptachlor epoxide have been tentatively accepted. In the Ontario survey commenced in 1967, the Ontario Department of Agriculture and Food, after consultation with the Department of National Health and Welfare and the Food and Drug Directorate, agreed to investigate all cases where the levels in milk fat were at or above 1.0 ppm DDT or lindane and 0.10 ppm dieldrin or heptachlor epoxide. Only 17 out of the 1,651 composite milk samples contained residues above the levels and these will receive further attention later in the paper.

Duggan, analyzing milk between 1963 and 1966, reported that milk fat across the United States contained a total of 0.227 ppm of chlorinated hydrocarbons (see table). The Ontario survey revealed an average total pesticide level of 0.171 ppm.

The total DDT in both surveys was the same. Dieldrin and lindane levels were lower in Ontario-produced milk than in USA milk, and heptachlor epoxide was significantly less in Ontario than USA milk. This may be a reflection of the climatic conditions in Ontario, which give rise to fewer insect problems, and hence require less insecticides than many areas in the United States. Virtually no milk in Ontario was free of DDT or dieldrin. On the other hand, only 7.6% and 3.0% of Ontario milk contained lindane and heptachlor epoxide, respectively. No samples were found containing methoxychlor. By "no residue" is meant "not detectable or below 5 ppb."

LEVEL OF CHLORINATED HYDROCARBONS IN MILK FAT

Insecticides	USA (1963-66) (ppm, in milkfat)	Ontario (1967-69) (ppm in milkfat)
DDT and metabolites	0.134	0.134
Dieldrin	0.043	0.031
Heptachlor epoxide	0.038	0.001
Lindane	0.011	0.005
Methoxychlor	0.001	0.000
TOTAL	0.227	0.171

A recent survey on the use of pesticides in agriculture, conducted by the Ontario Department of Health, revealed that, in 1968, 89% of the DDT and 85% of the aldrin and dieldrin were used in the 12 most southern counties of the province. Therefore, the residues in milk were compared on a regional basis. The province was divided into northern and southern halves, and the southern part was divided further into southern, western, central, and eastern regions, and the results were as follows:

RESIDUES IN MILK COMPARED ON A REGIONAL BASIS

Region	Number of samples	DDT and metabolites (ppm)	Lindane (ppm)	Dieldrin (ppm)	Heptachlor Epoxide (ppm)
Central	387	0.146	0.001	0.030	ND
Eastern	489	0.101	0.001	0.022	ND
Northern	70	0.062	ND	0.044	ND
Southern	372	0.193	0.002	0.043	less than 0.001
Western	333	0.114	0.017	0.017	0.003
Ontario	1,651	0.134	0.005	0.031	0.001

ND = not detected.

It can be noted that, while the DDT level in the southern region was the highest, it was only slightly higher than that in the central region and not quite twice that of the eastern or western region and about three times that of the northern region. It would therefore appear that, in spite of the more than 20-fold greater use pattern for DDT in the southern region, it was not reflected in the milk fat produced in that region, as might have been expected. Lindane and heptachlor epoxide were virtually absent from four of the five regions, and only occurred to any degree in the western region. Dieldrin was highest in the southern region, as might be expected, but this was only slightly higher than the level in the western region where 9% of the dieldrin was used in 1968. The use pattern of DDT, dieldrin, and aldrin was therefore not noticeably reflected in the milk produced in those areas, but appeared more uniformly distributed than might have been expected.

Where a composite sample from a tank truck, representing the shipment of milk from an average of 15 producers, contained residues in the fat above 1.0 ppm DDT or lindane, or 0.1 ppm dieldrin or heptachlor epoxide, samples of milk were collected from each producer for analysis.

From the 17 tank trucks rechecked, a total of 20 individual producers were found delivering milk above these levels. In five cases, the residues were between 0.10 and 0.20 ppm dieldrin. In these cases, the milk was resampled two weeks later and, since it was found to be declining, no further action was taken and the source of contamination was not determined. There were 12 producers with milk containing between 0.33 and 3.17 ppm dieldrin, one producer with 1.72 ppm lindane, and three producers with 3.36 to 17.7 ppm total DDT. In each of these cases, the producers were visited, and a total of 258 samples of feed, litter, water, spray, etc., were collected for analysis. In addition, 354 milk samples, including individual cow samples and composite herd samples, were collected for analysis.

In seven cases, the residues were the result of ingestion of contaminated feed or litter. In five cases, the cattle had been sprayed with an insecticide not registered for that purpose, or one that was contaminated with a persistent chlorinated hydrocarbon insecticide. In four cases, the source was no longer present and could not be determined. The following table summarizes these findings.

SOURCE OF CONTAMINATING INSECTICIDE

Method of contamination	Source of contamination	Number of cases	Contaminating Insecticide
By ingestion	Treated seed grains in feed	1	Aldrin, dieldrin
	Feed grains stored in contaminated bins	1	Aldrin
	Hay and feed grain low level contamination	1	Dieldrin
	Hay	2	Dieldrin
	Sweet corn silage	1	DDT, metabolites
	Sawdust litter	1	Dieldrin
By spraying or dusting	Contaminated spray	2	Aldrin, dieldrin
	Contaminated wettable powder used as dust	1	DDT
	Improper use	2	Lindane, DDT
Unknown	No source found	4	Dieldrin

The decline in total DDT and dieldrin varied according to the original level; however, once the source was removed, residues dropped rapidly at first and then slowly to reach acceptable levels in an average of 200 days. Lindane, on the other hand, dropped rapidly to acceptable levels in approximately ten days.

It might be added that the Ontario dairy farmer appeared to be extremely cautious in his use of insecticides around the farm and his

livestock. In none of the investigations involving dieldrin was it found that the farmer bought aldrin and dieldrin as such, and very few purchased treated seed for planting. In all except two cases, no direct blame could be placed on the producer for creating his problem.

The findings in this comprehensive survey on Ontario-produced milk should be reassuring to the consumers in the Province.

APPENDIX VI

Memo from the Ontario Pest Control Operators Association

To: The Pesticides Advisory Board

Submitted by: Ontario Pest Control Operators Association

Re: D.D.T. and matters pertaining to its usefulness, hazard, and necessity.

Sirs:

This association, being a highly active group, feels that, inasmuch as we deal largely with pest control problems in buildings, we have a special point of view with respect to pesticides.

Our services do not involve the application of chemicals to lands where food is grown, where birds and wildlife find sanctuary or where, in any way, the side-effects of our work will affect the level of pollution in the air, water or soil.

We apply our chemicals in or onto buildings, including homes, hospitals, factories and retail business.

Now there appears to be an effort to have one pesticide, namely D.D.T., banned for use in Ontario. These hearings are intended to help the Ontario Government determine a policy about the future use of, control of, or outright prohibition of D.D.T.

For many reasons we oppose such a ban, although the effects of a ban on D.D.T. would only affect a small portion of our work. A survey of our industry reveals that we use, on or in buildings, the following amounts of D.D.T.

10,000 lbs. of 50% powdered dust, or wettable powder, and

25,000 lbs. of 25% emulsifiable and/or oil soluble concentrate.

We feel that to switch *from* the liquid D.D.T. to a substitute chemical would represent neither an economic nor technical handicap. Therefore, our objection is not focused on this form of the chemical.

However, 50% D.D.T. as a tracking powder has been one of the safest, cleanest and most effective means of controlling rodents, namely mice, and it is also effective against bats.

To ban the sale of D.D.T. would mean that:

1. We would have to employ more toxic, more difficult to apply poisons to control rodents. This would then mean that complete re-evaluation of structural pest control licensing would be needed to ensure that the users of such more toxic chemicals were qualified to use them. We in the industry feel that, on average, most operators, having relied on anticoagulant baits and D.D.T. tracking dusts, would not immediately be fully versed in the use of arsenics, sulphates and phosphides, in the safest possible way. So in one way, banning D.D.T. would increase the public's exposure to the hazards of other more toxic rodenticides particularly in this field of our service.

2. We are of the opinion that while there have been notable instances of soil and water pollution by the *abuse* of the use of D.D.T., *none* of this pollution was caused by its use in the Structural Pest Control Industry. Structural pest control does not pollute. Our service is to provide a clean environment in the places where people live, work and find enjoyment.

3. From a philosophical point of view, our association harbours a fear that, if certain groups are successful in the banning of D.D.T., a precedent will be established whereby any pesticide, no matter how beneficial, no matter how difficult to substitute for, will be a target for groups whose numbers include persons militating against all pesticides in general.

In a world where the need for both increased food production, and just as importantly, in a world where our environment indoors and out, is striving for improvement, dignity and comfort, we must not restrict those necessary services which contribute highly to the attainment of either.

We propose:

A. That D.D.T. be continued as a pest control chemical for structural use.

B. That its use for land extermination be more carefully controlled through the Pesticide Control unit. This possibly could be done by requiring purchase registrations, investigation of inventories, and on-the-job surveillance of those operators known to have purchased D.D.T.

C. That the Pesticide Advisory Board, undertake as an independent government body, a public information program, telling the public about the merits of pesticides as well as their hazards. In addition, if the public knew how rigidly commercial pesticide applications are controlled, and what powers the Government has to control them, perhaps some of the doubts and fears of the public over the rising spectre, whether real or imagined, of pesticides poisoning our air, land and water, would, as it should, be put in a more balanced perspective.

We respectfully submit these suggestions and comments and we trust that from all these deliberations, a recommendation representing both concern and logic will result.

Yours very truly,
ONTARIO PEST CONTROL OPERATORS ASSOCIATION
B. P. Richardson,
Legal Committee

APPENDIX VII

Canada Department of Agriculture
Production and Marketing Branch
Plant Products Division

Pesticide Memorandum T-26
Ottawa, Ontario, June 11, 1969

Memorandum

Re: Proposed Revision of Uses for DDT Acceptable for Registration Under the Pest Control Products Act

Consistent with a policy of a continuing review of use patterns for pesticides registered under the Pest Control Product Act, DDT is being examined and a revised pattern of use is proposed. In preparing the review, the following points have been kept in mind:

1. There are certain pest problems where the use of DDT is essential in order to maintain production levels in agriculture.

2. For some uses, newer chemicals are available and have in practice replaced DDT.

3. Residues of DDT and its metabolites continue to be found in agricultural crops, as well as in animal and human body fats.

4. Some current farm management practices are not compatible with the use of DDT and facilitate the occurrence of residues.

5. An extended history of misuse in some pest control applications indicates the only way to eliminate such malpractices is to delete the recommendations from the list of accepted uses.

In the proposed use pattern, a copy of which is attached, the major deletions and changes are as follows:

1. Delete all uses on the vegetable crops asparagus, kale, parsnips, peppers and spinach.
2. Delete all uses on the field crops beans, corn and peas.
3. Delete all uses on forage crops.
4. Delete space spray and residual surface treatments in cattle barns and poultry houses.
5. Delete all uses on poultry.
6. Delete use on elm trees for control of elm bark beetles.
7. Delete space spray applications in dwellings.
8. Restrict applications in dwellings, non-food industrial plants and storage areas to surface treatments using dust or liquid formulations.
9. Delete application to humans for louse control. This should be considered as a medical treatment for use under a doctor's prescription.
10. Restrict operations against mosquitoes, black flies, and sand flies to adult control by ground application under provincial permit.

This proposed use pattern for DDT is being widely distributed to permit consideration and comment by all interested parties. Any criticism or valid objection which you may wish to make regarding this use pattern should be submitted to the Pesticide Unit by July 31, 1969. As soon after that date as possible, it is intended to establish the schedule of DDT uses which may be accepted for registration effective January 1, 1970.

ERH/ga

DDT

Common Name: DDT

Chemical Name: 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane

Formulations:

Du dust
EC emulsifiable concentrate
Gr granular
Sn solution
WP wettable powder

Guarantee in terms of: DDT

Classification: insecticide

PRECAUTIONARY LABELLING

Precautions: Avoid contact with skin or eyes. Avoid ingestion. Avoid contamination of food, feed, seed, drinking water and utensils. Do not spray in rooms containing pets, birds or tropical fish. Do not contaminate streams, lakes, ponds, irrigation water, water used by livestock or water used for domestic purposes. Use only for recommended purposes at correct rate. Keep in original container during storage. Destroy empty containers. Keep out of reach of children. Avoid use on food crops closer than 30 days of harvest unless otherwise noted.

First Aid: For external contact, wash skin with soap and water; rinse eyes with plenty of water. If swallowed, give a tablespoon of salt in warm water to induce vomiting. Repeat until vomit fluid is clear. Obtain medical attention.

Limitations:

1. Do not apply to crops within pre-harvest intervals specified below:
   7 days – tomatoes
   21 days – blueberries

30 days – cabbage, carrots, cherries, garlic, head lettuce, onions, peas, plums, potatoes, prunes, red beets, turnips.
35 days – cranberries
42 days – apples, apricots, grapes, peaches, pears, nectarines.

2. Do not apply after edible parts of crop begin to form.

3. Do not use on meat animals within 90 days of slaughter.

4. Do not use on dairy cattle at any time.

5. Do not feed treated crop or crop refuse to livestock at any time.

6. Do not feed treated tops to livestock.

7. Do not apply to young tomato plants or green bunching onions.

8. Do not apply after bunching starts.

9. Do not use on animals, fish, birds or plants.

10. Do not apply to ornamentals such as privet or viburnum. Use with caution on evergreens and Chinese elm.

11. Do not use on cats.

REGISTERED USES IN CANADA

Host or Location	Pest or Use	Dosage Rate (active ingredient) and Formulations	Directions for Use
apples, apricots, cherries, nectarines, peaches, pears, plums, prunes	apple leaf skeletonizer, apple maggot, lesser apple worm, apple red bug, codling moth, fruit tree leafroller, shot hole borer, peach tree borer, tussock moth, tent caterpillar, tarnished plant bug, canker worm, oriental fruit moth, Japanese beetle, thrips, leaf tiers, oyster shell scale, lecanium scale, curculio	32 oz. in 100 gal. of water WP, EC	Begin applications when insects are noted. Repeat at 10-14 day intervals or as required. Limitations (1) (5)
grapes	berry moth, leafhopper, flea beetles, canker worm, leafrollers, mealy bugs, plant bugs, thrips, leaf folder, tent caterpillars, Japanese beetle, leaf tier	16 oz. per acre Du, WP, EC	Apply to plants pre-bloom, post-bloom and 10 days later. Use 100 gal. of spray per acre. Limitation (1)

straw-berries	strawberry leaf roller, spittle bug, flea beetles, mealy bug, leafhopper, plant bugs, thrips, strawberry weevil, tarnished plant bug	32 oz. per acre Du, WP, EC	Begin treatment when first buds appear. Repeat as necessary. Limitation (2)
bush-berries (blue-berries, currants, goose-berries)	fruitworm, sawfly, leafhoppers	32 oz. per acre Du, WP, EC	Treat foliage as necessary for control. Limitation (2)
cranberries	leafhoppers	6 lbs. per acre Du, WP, EC	Apply as needed to control infestations. Limitation (1)
cane-berries (rasp-berries, black-berries, logan-berries, dewberries)	fruitworm, leafhopper, mealy bug, flea beetles, plant bugs, spittle bugs, thrips, leafroller, sawfly, stink bug, Japanese beetle	32 oz. per acre Du, WP, EC	Treat foliage as necessary for control. Limitation (2)
red beets	cutworms, tarnished plant bug, armyworm, leafhoppers, flea beetles, blister beetles	24 oz. per acre WP	Treat foliage when insects are noted. Limitations (1) (5)

REGISTERED USES IN CANADA

Host or Location	Pest or Use	Dosage Rate (active ingredient) and Formulations	Directions for Use
carrots	leafhoppers	32 oz. per acre Du, WP, EC	Use three applications at 10 day intervals in July and August. Not more than 3 applications per season. Limitations (1) (5)
	black swallow-tail butterfly	15-20 oz. per acre Du	Treat foliage as necessary to control infestations. Limitations (1) (5)
celery	tarnished plant bug, leafhoppers, loopers, cutworms, armyworms	32 oz. per acre Du, WP, EC	Apply when insects noted. Repeat as necessary for control. Treat soil around plants for control of cutworms. Limitation (8)
cole crops (broccoli, brussels sprouts, cauliflower, cabbage)	cabbage looper, flea beetles, imported cabbage worm, diamond back moth, climbing cutworms, red turnip beetle, purple-backed cabbage worm, thrips	32 oz. per acre Du, WP, EC	Treat when insects noted. Repeat as necessary. Limitations (1) (2)

eggplant, pepper	flea beetles, potato beetle, corn earworm, cucumber beetle, tomato hornworm, climbing cutworms	32 oz. per acre Du, WP, EC	Treat before edible parts have formed. Limitation (2)
leaf lettuce, head lettuce	leafhoppers, flea beetles, armyworm, cabbage looper, climbing cutworms, cabbage worm	32 oz. per acre Du, WP, EC	Apply to leaf lettuce in seedling stage only. Apply when insects noted. Limitation (1)
onions, garlic	thrips, armyworms, climbing cutworms	32 oz. per acre Du, WP, EC	Apply when first damage noted. Repeat at 7-10 day intervals during growing season. Limitations (1) (7)
potatoes, sweet potatoes	Colorado potato beetle, leafhoppers, thrips, potato psyllids, aphids, flea beetles, tarnished plant bug, tuber flea beetle	24 oz. per acre Du, WP, EC	Apply when insects noted. Repeat at 7-10 day intervals or as necessary. Limitations (1) (5)
radishes	flea beetles	24 oz. per acre WP	Apply to foliage at emergence.
sugar beets	armyworm, cutworms, blister beetle, flea beetles, lygus bug	16 oz. per acre Du, WP, EC	Apply to foliage for control of infestations. Limitation (6)

REGISTERED USES IN CANADA

Host or Location	Pest or Use	Dosage Rate (active ingredient) and Formulations	Directions for Use
tomatoes	tomato fruit-worm, climbing cutworms, corn earworm, blister beetles, stink bug, flea beetles, leaf-hoppers, army-worm, tomato hornworm	32 oz. per acre Du, WP, EC	Apply to plants as necessary. For cutworm control, treat soil near base of plants. For liquid treatments, use 100 gallons of spray material per acre. Limitations (1) (7)
turnips	flea beetle, red turnip beetle, caterpillars	32 oz. per acre Du, WP, EC	Apply to foliage as necessary to control infestations. Limitations (1) (6)
rape and mustard (seed crop only)	flea beetles	16 oz. per acre Du, WP, EC	Treat after plants have emerged and flea beetles are a problem. Do not apply after four leaf stage
tobacco	flea beetles	2 lbs. per acre Du, WP, EC	Treat when infestation is noted.
	cutworms	4 lbs. per acre WP, EC, Gr	Apply to plants and soil at time of transplanting or 7-10 days before transplanting.

greenhouse orna-mentals	crickets, sow-bugs, white fly, thrips, leaf-hoppers, midges, tarnished plant bug, cutworms, cabbage looper, leaf tiers, orchid scale, orchid weevil, diamondback moth, leafrollers, scale crawlers, tent caterpillars	5% dust Du 24 oz. in 100 gal. of water WP, EC	Apply at weekly intervals or as necessary to control infestations.
orna-mentals, trees, shrubs, flowers	tent cater-pillars, fall webworm, tussock moth, gypsy moth, canker worm, European pine shoot moth, armyworm, lace bug, aphids, thrips, leaf-hoppers, midges, May beetle, rose chafer, elm leaf beetle, Japanese beetle, diabrotica beetle, leaf tiers, sawfly, leafroller, yellowheaded spruce sawfly, taxus weevil, rose curculio, oyster shell scale, spruce budworm, codling moth, spittle bug, tree hoppers, tarnished plant bug, pine saw-flies, pine weevil	5-10% dusts at 32 oz. per acre Du 32 oz. per acre WP, EC	Apply treat-ments to foliage as needed to control infesta-tions of various pests. Repeat if necessary. Limitation (10)

REGISTERED USES IN CANADA

Host or Location	Pest or Use	Dosage Rate (active ingredient) and Formulations	Directions for Use
gladiolus, iris, and other bulbs or corms	thrips	3-10% dusts Du	Apply 1 oz. of dust per bushel of bulbs or corms before storage. During growing season, use a 5% dust at 7-10 day intervals.
lawns	armyworm, cutworm, webworm, chinch bugs, leafhoppers, earwigs, grassworms, leather-jackets (crane fly larvae)	2 oz. per 1000 sq. ft. in a water solution WP, EC	Apply dosage in 15-30 gal. of water to 1000 sq. ft. of lawn. Repeat as necessary for control.
vegetation and buildings on campsites and other outdoor areas	adult mosquitoes, black flies, sand flies	2-4 oz. per acre EC, Sn	**For ground application:** By provincial permit only.
pet animal quarters	fleas	5% solution Sn	Treat bedding, floors, etc. Repeat as necessary. Limitation (9)

dwellings, non-food industrial plants and storage areas	silverfish, crickets, fleas, lice, spiders, centipedes, earwigs, bedbugs, clothes moths, carpet beetles, gnats, mosquitoes, midges, stored product insects	10% dust Du 5% solution WP, EC	Dust or spray lightly on surfaces where insects occur. Repeat as necessary. Use spray at a rate of 1 gal. per 1000-1200 sq. ft. Treat under carpets, on base-boards, mattres-ses, and bedframes as required by type of pest to be controlled. Limitation (9)
woolen fabrics, clothing, rugs, carpets, blankets	clothes moths, carpet beetles	10% dust Du 1-5% solution Sn, EC	Dust lightly on fabrics before storage. Spray on rugs and carpets to obtain 0.5% deposit by weight. For fabrics, blankets or garments, treat to obtain 0.3% deposit by weight. Protects for one season.
beef cattle	fleas, lice, ticks, horn flies, gnats	10% dust Du 0.5% solution WP, EC	Dust onto animals to control infesta-tions or to protect cattle from pests. Spray solution on cattle at rate or 1-2 gallons per animal for lice. Use 2-4

REGISTERED USES IN CANADA

Host or Location	Pest or Use	Dosage Rate (active ingredient) and Formulations	Directions for Use
			qts. per animal for other pests. Repeat as necessary. Limitations (3) (4)
		5% oil solution Sn, EC	Apply to backrubbers. Keep applicators well supplied. Limitations (3) (4)
sheep, goats	ticks, lice, fleas	10% dust Du 0.5% solution EC	Dust or spray at rate of 2 qts. per animal. Repeat as necessary. Limitation (3)
hogs	lice	10% dust Du 0.5% solution WP, EC	Dust or spray animals to control infestations. Use 1-2 qts. of spray per animal at 2 week intervals. Limitation (3)
horses	fleas, ticks, lice	10% dust Du	Apply treatment to animals to control infestations. Repeat as necessary.

dogs, foxes	fleas, lice, ticks	10% dust Du	Dust on coat and work down to skin. Repeat as necessary. Limitation (11)

APPENDIX VIII

THE PESTICIDES ACT, 1967

O. Reg. 386/69.
General.
Made—September 17th, 1969.
Approved—September 18th, 1969.
Filed—September 24th, 1969.

REGULATION MADE UNDER THE PESTICIDES ACT, 1967

1. Ontario Regulation 445/67, as amended by Ontario Regulations 189/68, 139/69, 197/69 and 222/69, is further amended by adding thereto the following sections:

22b.—(1) Notwithstanding any other provisions of this Regulation or the provisions of any other Act or Regulation, no person shall use DDT or TDE except,

(a) for the purpose of bat extermination while holding a licence to perform structural exterminations;

(b) for cut worm control on tobacco, except from an airborne machine or concentrated airblast machine; or

(c) for plant bug extermination on apples, provided an official of the Department of Agriculture and Food verifies that a plant bug situation exists.

(2) No person shall perform an extermination under clause *a, b* or *c* of subsection 1 unless he has obtained a permit in duplicate therefor in Form 16 from the Director.

(3) The Director may refuse to issue a permit in Form 16 where he is of the opinion that the extermination in respect of which the permit is sought cannot be carried out in safety.

22c. Notwithstanding the provisions of section *22b*, the Minister may grant permission to use DDT or TDE where in his opinion an emergency has arisen or the public interest so dictates.

22d. —(1) No person shall sell or distribute DDT or TDE to any other person for the purpose of performing an extermination except where the purchaser presents the original and one copy of a duly signed permit in Form 16.

(2) The vendor shall retain the duplicate of the permit.

(3) The vendor shall return the duplicate permit to the Department at the end of each month showing how much DDT or TDE was purchased on the permit.

22e. Every exterminator shall report to the Department not later than the 1st day of October in each calendar year, the disposition of all DDT or TDE purchased by him during the preceding twelve-month period.

2. Ontario Regulation 445/67, as amended by Ontario Regulations 189/68, 139/69, 197/69 and 222/69, is further amended by adding thereto the following Form:

FORM 16

The Pesticides Act, 1967

PERMIT TO PURCHASE AND USE DDT
OR TDE

Permission is hereby granted to ....
(name of exterminator)

to purchase and use .... of ....
(amount) (substance)

for the control of .... on the premises
(name of pest)

at ....
(address of premises)

Date ...., 19 ...

....
(Director)

3. This Regulation comes into force on the 1st day of January, 1970.

THOMAS L. WELLS
Minister of Health

Dated at Toronto, this 17th day of September, 1969.

(2543) 40